DOES NOT CIRCULATE

UNIVERSITY OF MICHIGAN
JUL 27 1955
MEDICAL

# THE ANNALS

## OF OTOLOGY RHINOLOGY & LARYNGOLOGY

ALS PUBLISHING COMPANY — SAINT LOUIS 1, MISSOURI

ME LXIV JUNE, 1955 NUMBER 2

THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

Published Quarterly by

THE ANNALS PUBLISHING COMPANY, St. Louis, 1, U. S. A.

Entered at the Postoffice, St. Louis, Mo., as Second-class Matter.

THE ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY is published quarterly by The Annals Publishing Company, P. O. Box 1345, Central Station, St. Louis 1, Missouri. Subscriptions and all communications of a business nature should be sent to this address. Manuscripts for publication should be sent to 1010 Beaumont Building, St. Louis, 8, Missouri.

The subscription price in United States and all foreign countries is $12.00 per annum payable in advance. Single copies may be had at the rate of $3.25 each. Unless otherwise specified, subscriptions will begin with the current number.

In notifying this office of change of address, both the old and the new address should be given.

EDITORIAL OFFICE — 1010 BEAUMONT BUILDING, 8

BUSINESS OFFICE — P. O. BOX 1345, CENTRAL STATION, 1

Information for contributors will be found on the inside back cover.

# ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED IN 1892

VOLUME 64

**Editor**

ARTHUR W. PROETZ, M.D.
Beaumont Building, St. Louis, 8

**Associate Editor**

BERNARD J. MCMAHON, M.D.
Forsyth Building, St. Louis 24

**Editorial Board**

L. R. BOIES, M.D. . . . . *Minneapolis*
LOUIS H. CLERF, M.D. . . . *Philadelphia*
SAMUEL J. CROWE, M.D. . . . *Baltimore*
EDMUND P. FOWLER, JR., M.D., *New York*
DAVID R. HIGBEE, M.D. . *San Diego, Calif.*
ANDERSON C. HILDING, M.D. . . *Duluth*
FREDERICK T. HILL, M.D. . *Waterville Me.*
HAROLD I. LILLIE, M.D. . *Rochester, Minn.*
JOHN G. MCLAURIN, M.D. . . . . *Dallas*
LEROY A. SCHALL, M.D. . . . . *Boston*
BEN H. SENTURIA, M.D. . . . . *St. Louis*
FRANCIS A. SOOY, M.D. . . *San Francisco*
H. MARSHALL TAYLOR, M.D., *Jacksonville, Fla.*
O. E. VAN ALYEA, M.D. . . . . *Chicago*

*Published Quarterly*

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE . 1010 BEAUMONT BUILDING, 8
BUSINESS OFFICE . P. O. BOX 1345, CENTRAL STATION, 1

ST. LOUIS, MO., U.S.A.

COPYRIGHT, 1955
ANNALS PUBLISHING COMPANY

Annual Subscription in United States and All Foreign Countries is $12.00 in Advance.

# Contents.

PAGE

XXXVI—Indications for the Use of the Direct Laryngoscope for Introduction of the Bronchoscope. Chevalier Jackson, M.D., Schwenkville, Pa. 345

XXXVII—Some Facts and Fancies about the Utricle. W. J. McNally, M.D., Montreal, Canada 355

XXXVIII—Transparent Reconstruction Models. V. E. Negus, F.R.C.S., London, Eng. 364

XXXIX—The Role of the Cerebrum in Hearing. John E. Bordley, M.D. and Harriet L. Haskins, A.M., Baltimore, Md. 370

XL—The Maximum Strength of the Tympanic Muscles. Ernest Glen Wever, Ph.D., Jack A. Vernon, Ph.D., and Merle Lawrence, Ph.D., Princeton, N. J. 383

XLI—Combined Effect of Sound and Oxygen Deprivation upon Cochlear Microphonics in Guinea Pigs. Juergen Tonndorf, M.D., Reed W. Hyde, Captain, USAF (MC), and F. A. Brogan, B.A., Randolph Field, Texas 392

XLII—Reports on the Chemical Composition of the Fluids of the Labyrinth. IV. Flow Elasticity and Flow Potentials in Peri- and Endolymph from Sharks. Thure Vilstrup, M.D., C. E. Jensen, M.S., and Jörgen Koefoed, M.S., Copenhagen, Denmark 406

XLIII—Observations and Considerations on Cigarette Smoking. Mervin C. Myerson, M.D., Beverly Hills, Calif. 412

XLIV—Plastic Esophageal Tube. William W. Montgomery, M.D., Boston, Mass. 418

XLV—The Coexistence of Malignant Tumors in the Trachea and a Distal Secondary Bronchus. Donald S. Bolstad, M.D., Detroit, Mich. 422

XLVI—A Reference Point System for Interpreting Vestibular Tests. A. M. Paisley, M.D., Jacksonville, Ill. 425

XLVII—Pneumatocele of the Temporal Bone. George Botros, M.D., Cairo, Egypt 436

XLVIII—Vocal Technique for Treatment of Certain Speech Difficulties. Richard O. Bullis, M.D. and Alexander Kisselburgh, Los Angeles, Calif. 445

**The Scientific Papers of the American Laryngological Association**

XLIX—Evaluation of Dissection of the Neck in Carcinoma of the Larynx. Louis H. Clerf, M.D., St. Petersburg, Fla. 451

L—Mixed Tumors of the Posterior Pharyngeal Wall. Fred Z. Havens, M.D. and Lewell C. Butler, M.D., Rochester, Minnesota 457

LI—Vasomotor Rhinitis. Theo. E. Walsh, M.D., St. Louis, Mo. 466

LII—The Radical Obliterative Frontal Sinus Operation. A Consideration of Technical Factors in Difficult Cases. Robert L. Goodale, M.D., Boston, Mass. 470

## CONTENTS—Continued


PAGE

LIII—Unilateral Vocal Cord Paralysis. Daniel S. Cunning, M.D., New York, N. Y. ..... 487

LIV—General Tonsillectomy. Suggestions for Management and Peritonsillar Injection. Paul M. Moore, M.D., Cleveland, Ohio ..... 494

**The Scientific Papers of the American Otological Society**

LV—Auditory Fatigue and Adaptation in the Differential Diagnosis of End-Organ Disease. J. D. Hood, London, England ..... 507

LVI—An Additional Five Year Review of Some Cases of Vertigo Reported in 1949. W. J. McNally, M.D. and E. A. Stuart, M.D., Montreal, Canada ..... 519

LVII—Elastic Tissue of the Eustachian Tube. Stacy R. Guild, Ph.D., Baltimore, Md. ..... 537

LVIII—Chemodectomas of the Glomus Jugulare (Nonchromaffin Paragangliomas) with Especial Reference to their Response to Roentgen Therapy. Henry L. Williams, M.D., Donald S. Childs, Jr., M.D., Edith M. Parkhill, M.D. and David G. Pugh, M.D., Rochester, Minnesota ..... 546

LIX—A Review of Some Recent Studies Concerned with the External Ear. B. H. Senturia, M.D., L. H. Sophian, M.D., S. P. Chiang, Ph.D., O. H. Lowry, M.D., St. Louis, Mo. ..... 567

**The Scientific Papers of the American Broncho-Esophagological Association**

LX—Electrocardiographic Studies During Bronchoscopy in Rated-Risk Cardiac Patients. A Preliminary Report. Walter H. Maloney, M.D. and Franklin C. Massey, M.D., Philadelphia, Pa. ..... 580

LXI—Observations on Aqueous and Oily Media in Lungs of Experimental Animals. L. C. Smith, M.D. and James A. Harrill, M.D., Winston-Salem, N. C. ..... 588

LXII—Esophageal Varices in Hepatic Cirrhosis: An Endoscopic Study. Stanton A. Friedberg, M.D., Chicago, Hugh D. Bennett, M.D., Houston, Texas, Harkishen Singh, M.D. and John Lindberg, M.D., Hines, Illinois ..... 599

**Clinical Notes**

LXIII—Syphilis and Oral Cancer. S. Gordon Castigliano, M.D., Philadelphia, Pa. ..... 608

LXIV—The Effect of Salicylates on the Inner Ear. Jules G. Waltner, M.D., New York, N. Y. ..... 617

**Society Proceedings**

Chicago Laryngological and Otological Society, Meeting of Monday, March 1, 1954. Otolaryngologic and Rhinologic Problems of Today—Recording of Vestibular Nystagmus—"Filmettes" As A Teaching Aid In Otolaryngology: Motion Picture ..... 623

Meeting of Monday, April 5, 1954. Morphologic Changes In the Mastoid Bone Under Antibiotic Therapy—Changing Concepts in Rhinoplastic Surgery ..... 629

**Abstracts of Current Articles** ..... 634

**Books Received** ..... 658

**Notices** ..... 660

**Officers of the National Otolaryngological Societies** ..... 664

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 64 JUNE, 1955 No. 2

XXXVI

# INDICATIONS FOR THE USE OF THE DIRECT LARYNGOSCOPE FOR INTRODUCTION OF THE BRONCHOSCOPE

CHEVALIER JACKSON, M.D.
SCHWENKVILLE, PA.

The laryngoscope is not necessary for the introduction of the bronchoscope in any case, and it is not used by experienced bronchoesophagologists for routine bronchoscopies. The fact that they use it occasionally implies that its use is considered advantageous in some cases. An effort to formulate these implied special indications is made herein.

*Imminent Asphyxia.* It is one of the aphorisms of the bronchoscopic clinic that "death is an irreversible process; imminent asphyxia is not." Imminent asphyxia is an extremely important as well as an extremely urgent indication for laryngoscopic bronchoscopy. For life-saving efficiency nothing equals the prompt, smooth, skillful laryngoscopic exposure of the laryngeal lumen and the insertion of the bronchoscope through it. In the terminal phase of asphyxia due to any cause, the tongue drops backward and occludes the laryngeal orifice. The laryngoscope instantly eliminates this lingual death zone.[1] If an obstruction is encountered, it can be removed. If there is no obstruction, its absence is known with certainty. With the bronchoscope in position, tubal access to the pulmonary alveoli

From the Department of Laryngology and Bronchoesophagology, Temple University School of Medicine, Philadelphia, Pa.

is afforded for the insufflation of oxygen and vaporized cardiac stimulants. So far as known today the only practical channel for getting oxygen into the blood is through the alveolar walls. Without an adequate supply of oxygenated blood the heart muscle stops. With the bronchoscope in position, if respiration has also ceased, oxygen and vaporized stimulants, such as the ammoniacal group, can be "pumped" into the alveoli by artificial respiration. Of all methods the best for this purpose is that of Sylvester, because it does not interfere with efficient use of the bronchoscope as a channel for the life-saving vapors.[2] As stated by Jackson in 1922,[3] every interne should be taught and skilled in the prompt exposure of the glottis and the introduction of the bronchoscope through it, for this life-saving work.

In the foregoing formulation of the indications for laryngoscopic bronchoscopy there is no implication of a contraindication to tracheotomy. When by examination with tube or mirror, laryngeal obstruction is known to exist and the cardinal signs of obstructive laryngeal dyspnea (indrawing at the suprasternal notch, around the medial third of the clavicles and at the epigastrium) are present, tracheotomy is urgently indicated in the absence of endoscopic instruments and a trained peroral endoscopist. In such cases of extremely urgent dyspnea it is a life-saving procedure.

It is unnecessary here to enumerate the many causes of imminent asphyxia. In fact, in many cases the cause is unknown until revealed by laryngoscopic bronchoscopy, and it is in these cases that the use of the laryngoscope is of utmost importance. To clear the airway from the dorsum of the tongue to the segmental bronchi is the primary indication and it should be done instantly. This procedure is indicated in all forms of respiratory arrest, such as electric shock, inhalation of carbon monoxide and other lethal gases, bulbar lesions or trauma, bilateral pneumothorax, tight tucking of sleeping babies so they cannot turn over.[4] In cases of seeming *drownage from submersion* laryngoscopic bronchoscopy is urgently indicated.[4] After aspiration of fluid the oxygen tube is attached to the proximal inlet of the bronchoscope and the oxygen-laden air is "pumped" into the lungs by artificial respiration by the Sylvester method. If the oxygen is insufflated through an aspirating tube, the tube must be of small diameter to permit an ample return flow, and the flow of oxygen must be gentle. Otherwise the excessive pressure may obliterate the blood vessels in the vesicular walls[2] This precaution is absolutely necessary in insufflation of any vapor for any purpose.

It may be thought that imminent asphyxia is rare in the experience of any one physician; but Paluel J. Flagg, President, and for

many years Executive Secretary of the Society for the Prevention of Asphyxial Death, has made the statement that, as a broad general estimate, the average annual mortality by asphyxia in the United States, may be said to be "in excess of 50,000."[8]

*Asphyxia Neonatorum.* Imminent asphyxia neonatorum is an extremely important indication for use of the laryngoscope and the prompt passing of the bronchoscope through it. As stated by Jackson and Jackson,[4] "The highest human death rate in vital statistics is in the first fifteen minutes after birth." The mortality rate in this short period is over two thousands times greater than that of any other period of human life.[8] Obstetricians, by their good work, have done much to bring the neonatal mortality rate down to the present reduced level. As stated by the just quoted authors,[5,9] further reduction of this deplorable mortality awaits the establishment in every obstetric ward, of trained personnel and equipment for instant performance of laryngoscopic bronchoscopy. As shown by the just mentioned authors, a large proportion of these deaths are due to obstructions of the air passages removable by direct means. All human beings are born with both lungs drowned in meconium laden amniotic fluid. There is no air below the larynx with which to create a bechic blast.[4,7] The only natural spontaneous means of clearing the neonatal airway is by compression of the lungs by the thoracic muscles in a way mechanically similar to what later becomes the "tussive squeeze," (Jackson and Jackson[6,7]). If the thoracic muscular action is too weak the infant drowns in meconium laden amniotic fluid. As physical and roentgen examinations show no air in the lungs many of such cases are recorded as stillbirths. Of course, in most cases the fluid can be removed by laryngoscopic aspiration with a silk woven aspirating tube, but the particular indication for the laryngoscopic bronchoscopy is that, in some cases, there is laryngeal stenosis. Seconds count, and it is advisable to have the infant bronchoscope in the sterile-instrument nurse's hands ready for instant insertion through the stenosed larynx.

Chevalier Jackson[10] records numerous observations of neonatal anomalies. He illustrates cases of laryngeal obstruction with imminent asphyxia neonatorum due to partial absence of a larynx, laryngeal clefts, fistulae, chondromalacia, webs, diaphragms, teratoma, bilateral midline cordal paralysis. In the cases of bilateral midline paralysis, the condition was attributed by the attending obstetrician to pressure of misplaced obstetric forceps, because complete spontaneous recovery soon followed the emergency laryngoscopic bronchoscopy. In all of the observed cases of anomalies, except those

of partial absence of the larynx, the insertion of the bronchoscope was feasible. In the cases of web and those of complete diaphragm the obstruction yielded to precisely directed axial advance of the infant bronchoscope. This precision was easily accomplished with the aid of the laryngoscope and the proper position in which the head was held. Aspiration of fluid was instantly followed by gentle administration of oxygen through the proximal inlet of the bronchoscope, aided by gentle artificial respiration by the Sylvester technique. Prolonged laryngoscopic treatment after the emergency was required for cure of the laryngeal stenosis in the case of anomalous stenosis.[10]

*Laryngoscopic Bronchoscopy in Children.* In the decision as to the use of the laryngoscope for insertion of the bronchoscope in children, certain special factors are to be considered. In children too young for satisfactory preliminary laryngeal examination with the mirror, the larynx should be examined with the direct laryngoscope, because the bronchoscope is not so satisfactory for this purpose. With the larynx exposed by the laryngoscope the simplest thing to do is to insert the bronchoscope through it. In all laryngeal examinations in which bronchoscopy is not contemplated the bronchoscope nevertheless should be in the hands of the instrument nurse for prompt insertion if needed. In cases of croupy cough intubation tubes and instruments for laryngoscopic intubation should be on the sterile instrument table.

In children, *any laryngeal sound or symptom* is an indication for laryngoscopic bronchoscopy. To omit bronchoscopy in such cases would risk missing important diagnostic information. This would be true even if the laryngoscopic examination were negative, or if the laryngeal sound or symptom should disappear with the laryngoscope in the examining position, as it usually does in cases of congenital laryngeal stridor, for example. Moreover, in any kind of case, with the laryngoscope in place exposing the glottis, as just mentioned, the simplest thing to do is to insert the bronchoscope through it and complete the examination at the one seance.

In the *initial diagnostic bronchoscopy* in children, some bronchoscopists prefer to use the laryngoscope for introduction of the bronchoscope, and then decide whether to use it or not in subsequent bronchoscopies, if any should be needed. Other bronchoscopists prefer to do all bronchoscopies in children, *initial* as well as *routine*, without the laryngoscope.

In children with a *foreign body in the tracheobronchial tree*, laryngoscopic bronchoscopy may be indicated for accurate determination as to the presence of subglottic edema, and, if present, the degree.

This condition can be more accurately determined with the laryngoscope than with the bronchoscope.

All of these questions of procedure are for decision by the experienced operator, who will and should, of course, include consideration of his own equation of skill along with other phases of technique.

*Laboratory Specimens.* When laboratory specimens are desired for research, skillful laryngoscopic bronchoscopy will provide the means of taking specimens absolutely uncontaminated by the abundant flora of lingual, faucial, tonsillar and pharyngeal regions. The primary purpose of the laryngoscope for insertion of the bronchoscope was to prevent carrying such materials into the tracheobronchial tree. After the revelations of bronchoscopy in foreign-body cases demonstrated the powerful resistance of the bronchial walls to transmural infection,[5] the protection of the laryngoscope against the infections carried by the patient, was considered unnecessary. This inference has been supported by the clinical fact that no case of infection justly attributed to bronchoscopy without the laryngoscope has yet been reported in the available literature. The relative immunity of the carrier to his infections does not warrant any relaxation of sterile discipline in the operating room, but it does justify the insertion of the bronchoscope without the protection of the laryngoscope.[4]

*Acquiring Skill.* All bronchoesophagologists agree that an important indication for laryngoscopic introduction of the bronchoscope is to develop skill in introducing the bronchoscope without the laryngoscope. To begin bronchoscopic work by attempts to find the glottis with the bronchoscope, usually results in a clumsy prolonged search that is an ordeal for the patient and a disappointment for the operator, as well as a discredit to bronchoscopy. Simply stated, the pharynx is a large vestibular cavity at the parting of the way for the food and air on which human life depends for its supplies. The apertures of these two passage ways are obscured by the crowding in of bulk tissues activated by powerful lingual and pharyngeal muscles. To find the glottis with a bronchoscope promptly and with the minimum of discomfort to the patient, a thorough knowledge of the topographic anatomy and endoscopic appearance of the entire region is necessary. The first step in acquiring this knowledge is many hours of laryngoscopic and bronchoscopic study of numerous cadavers of various ages of life. This is basic, but it is not enough. The next step is an abundant clinical experience in laryngoscopic bronchoscopy. Then routine bronchoscopy can be done quickly, safely and with little discomfort to the patient. The laryngoscope will be used rarely

and only when indicated in the opinion of the bronchoscopist in the particular case.[4]

*Current Practice.* With a view to obtaining information on this phase of the subject a request for data was sent to eight of the bronchoesophagologic clinics of Philadelphia. Parenthetically it may be stated that the distally lighted bronchoscope is used at all of these clinics. It should also be stated that there is no invidious distinction implicit in the presentation of only Philadelphia clinics. There are now so many large bronchoesophagologic clinics in the United States, that to send a questionnaire to all of them and present the replies, would expand this presentation to the proportions of a large monograph. From the reliable reports and observations available as to current practice in many of the elsewhere located clinics it is evident that current practice is similar to that of the Philadelphia clinics.

A clear statement of current practice in Philadelphia is the response of Dr. Louis H. Clerf of the Jefferson Medical College Hospital bronchoesophagologic clinic. It is as follows: "I have been introducing the bronchoscope without the laryngoscope in all cases possible since 1930. It is difficult to employ this method in very young children and infants, and as you indicated in your letter in cases of impending asphyxiation. These are practically the only exceptions. I have found that it is much easier on both the operator and the patient if the laryngoscope is not used. I have, however, made it a routine practice in the training of residents to have them start bronchoscopy using the laryngoscope for introduction. This is desirable because of their lack of knowledge of the gross appearances of the larynx through an electrically lighted tube and gives them a better insight into the problem as a whole. I wean them, however, as soon as they begin to know their way about."

The current practice at the bronchoesophagologic clinic of the Graduate Hospital of the University of Pennsylvania is given by Dr. Gabriel Tucker as follows: "I have usually carried out the routine of direct laryngoscopy before inserting the bronchoscope particularly on the first examination; on subsequent examinations we have not used the laryngoscope for the insertion of the bronchoscope. In very difficult cases where there are projecting upper teeth we have found it easier for the patient to omit the use of the laryngoscope. In children under six years of age we have passed the bronchoscope through a laryngoscope. I think I can say that we follow the same procedure as they do in the Temple Clinic."

The current practice at the clinic of the University of Pennsylvania Hospital and the Children's Hospital of Philadelphia is

summarized by Joseph P. Atkins as follows: "It is the practice in our clinics to use a laryngoscope for the introduction of a bronchoscope in small children as a routine; in adults when direct visualization of the larynx constitutes a necessary part of the investigation of the patient; and in circumstances of impending asphyxia. We do not routinely use a laryngoscope for the introduction at other times. An exception to this situation exists in the case of residents in the beginning of their training. In order to acquaint them with the use of the laryngoscope and to develop facility, they routinely use the laryngoscope for the introduction of the bronchoscope. When they have mastered this technique, they are then encouraged to do bronchoscopy in adults without the use of the laryngoscope. In the teaching of bronchoesophagology at the Graduate School of Medicine we have felt that the time exists to teach only one method of bronchoscopy and they, therefore, are limited to instruction using the method that is universally applicable. At the same time, however, numerous demonstrations are given in which the other technique is used and the reason for limiting practical instruction to the introduction of the laryngoscope is explained to the students. I believe that anyone who is familiar with the technique of esophagoscopy and who has been trained in the introduction of bronchoscope through the laryngoscope, is in a position to understand both methods."

Dr. Emily Lois Van Loon reports on the current practice at the bronchoesophagologic clinics of the Women's Medical College of Pennsylvania and the St. Christopher's Hospital for Children, as follows: "In our clinics we still use the laryngoscope for introducing the bronchocsope in all infants and children; we use it to introduce the bronchoscope in about one-third of the adults. It is my feeling that speed in introduction of the bronchoscope minimizes the patient's discomfort; therefore, in uncooperative patients, or in individuals who have a narrow epiglottis, I believe that the bronchoscope can be introduced more quickly through the laryngoscope than without it."

Dr. F. Johnson Putney of the Jefferson Medical College Hospital Clinic reports current practice as follows: "I have used the bronchoscope alone for many years. Its routine use without the laryngoscope proved quite satisfactory and I see no advantage and many disadvantages to using the laryngoscope before introducing the bronchoscope. The laryngoscope is employed only in the occasional instance where there is a second laryngeal lesion in which biopsy or removal of tissue is required. Secondly, in teaching residents I require them to first become familiar with the technique of exposing the larynx with a laryngoscope, followed by introduction of the

bronchoscope allowing them to use the bronchoscope alone. As you know, a man just beginning endoscopy has difficulty in finding the larynx, and the laryngoscope with its larger caliber makes this easier for the beginner. In all other instances the bronchoscope is used routinely for examination and treatment."

Dr. Chevalier L. Jackson states that at the bronchoesophagologic clinic of Temple University Hospital the bronchoscope is inserted in all routine cases without preliminary direct laryngoscopic exposure of the larynx. The laryngoscope is used only in the case of imminent asphyxia and in children too young for satisfactory preliminary mirror examination of the larynx.

Dr. Charles M. Norris of this clinic kindly kept a record of 200 consecutive cases of bronchoscopy and noted that the laryngoscope was used for insertion of the bronchoscope in only 9 of the 200 cases of bronchoscopy. Of the 9 cases 7 were in children.

Current practice at the bronchoscopic clinic of the Hahnemann Medical College Hospital is given by Dr. Walter H. Maloney as follows: "It is routine in my clinic to introduce the Jackson bronchoscope directly without the use of the laryngoscope in all cases where the co-operation of the patient can be obtained and where there is no anatomical deformity preventing such a technique. With an experienced operator we reserve the use of the laryngoscope for introduction to the cases of young infants and true emergencies where there is not time to waste in trying the other technique first. I do instruct the beginning student in endoscopy in the technique of introduction of the bronchoscope with the laryngoscope first and as soon as he has grasped these fundamentals he is instructed in the introduction of the bronchoscope without the laryngoscope. I certainly agree that it is an erroneous statement that the distally lighted bronchoscope requires a laryngoscope for its introduction unless that statement were qualified as I have indicated above and as indicated in your letter to me."

Current practice at the bronchoscopic clinics of Pennsylvania and Presbyterian Hospitals is reported by Dr. William A. Lell as follows: "Personally, I find that the proper exposure of the larynx by using the laryngoscope affords an added measure of safety during the process of passing the bronchoscope through the glottis, since the structures can be visualized clearly and trauma of the laryngeal structures is more easily avoided than by the introduction of the bronchoscope without laryngoscopic aid. I find this to be particularly true in individuals who persist in swallowing or have considerable secretions in the pharynx. The only time that I utilize the

passage of the bronchoscope without the laryngoscopic aid is in patients with whom the procedure is repeated and I have a fair knowledge of their behavior. In children, of course, I never think of passing the bronchoscope without the preliminary exposure of the larynx by using the laryngoscope."

Dr. George L. Whelan of the Philadelphia General Hospital gives the following statement of current practice: "In the majority of routine adult cases the bronchoscope is inserted without preliminary direct laryngoscopic exposure of the larynx. The laryngoscope is not infrequently used on patients with tuberculosis before introducing the bronchoscope when the patient is being studied for the first time, so that we may visualize the larynx for a moment before introducing the bronchoscope. Sometimes in training residents the larynx is exposed by the operator and the resident then introduces the bronchoscope through the laryngoscope, with the thought in mind that it may help the resident to familiarize himself with the important landmarks."

SUMMARY

The laryngoscope is not necessary for the insertion of the distally lighted standard bronchoscope in any case, and experienced bronchoesophagologists do not use it for routine bronchoscopies; but the fact that they occasionally use it implies that there are particular cases in which its use for this purpose is advantageous. An effort is made in foregoing pages to formulate the implied indications for its use.

Old Sunrise Mills.

REFERENCES

1. Jackson, Chevalier, and Jackson, Chevalier L.: The Lingual Death Zone in Asphyxia, N. Y. State J. Med., 34:1-4 (Aug. 1) 1934.

2. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope, 1915.

3. Jackson, Chevalier: Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Co., 1922.

4. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoesophagology, Philadelphia, W. B. Saunders Co., 1950.

5. Jackson, Chevalier: Observations on the Pathology of Foreign Bodies in the Air and Food Passages, Muetter Lect. Surg. Gyn. & Obst., 28-201 (March) 1919.

6. Jackson, Chevalier: Bronchial Obstruction, J. Dis. Chest, 17:125 (Feb.) 1950.

7. Jackson, Chevalier, and Jackson, Chevalier L.: Peroral Pulmonary Drainage, Natural and Therapeutic, with Especial Reference to the "Tussive Squeeze," A. J. Med. Sci., 186:849 (Dec.) 1933.

8. Flagg, Paluel J.: Art of Resuscitation, New York, Reinhold Publishing Co., pp. 30, 31, 1944.

9. Jackson, Chevalier, and Jackson, Chevalier L.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945.

10. Jackson, Chevalier: Anomalies of the Larynx in Coates, George M., and Schenck, Harry P., Practice of Otolaryngology, Hagerstown, Md., W. F. Prior Inc., 1955.

XXXVII

# SOME FACTS AND FANCIES ABOUT THE UTRICLE

W. J. McNALLY, M.D.

MONTREAL, CANADA

The internal ear was called a labyrinth because its anatomy is so complex and its function so mysterious. As long ago as 1891 Breuer[1] distinguished between the functions of the semicircular canals and of the otoliths yet much of what we know about the interaction of these two parts of the internal ear is still speculative and not well supported by experimental proof. Breuer stated that, whereas the semicircular canals are stimulated by rotation or angular acceleration, the otoliths are stimulated by linear acceleration and by change of position of the head in space in relation to gravity. He gave to the otoliths the name of 'static sense organs.'

Kreidl[8] performed an ingenious experiment in 1893 which attracted widespread interest. After a decapod crustacaean Palaemon had moulted and therefore had lost its otoliths, he placed the young animal in a dish containing magnetic sand and the animal, as was its habit, placed a bit of the environmental sand in its otocyst. Kreidl found that by bringing a magnet to the animal he could make it assume forced positions by moving the magnetized sand to different parts of the otocist. This led to the assumption that it is the pressure of the otolith upon the underlying hair cells of the otocyst or macula that is the stimulus which brings about readjustment of posture in an animal in response to a change of the head which alters its relation to gravity. Quix and Magnus[12,15] disagreed as to whether it was pressure or pull of the overlying otolith on the macula which is the adequate stimulus of an otolithic end organ. The matter is still unsettled.

In the lower animals there are a number of otolithic organs such as the utricle, the saccule, the lagena, the macula neglecta, the pars basilaris etc. In man there are only two, the utricle and the saccule. My remarks will be limited, for the most part, to the utricle because the majority of experiments indicate that the saccule is not stimulated by motion or change of position but probably by vibrational stimuli.

---

This paper was written as part of the memorial tribute to Dr. Harris P. Mosher, but by inadvertence was received too late for inclusion in the Memorial number.

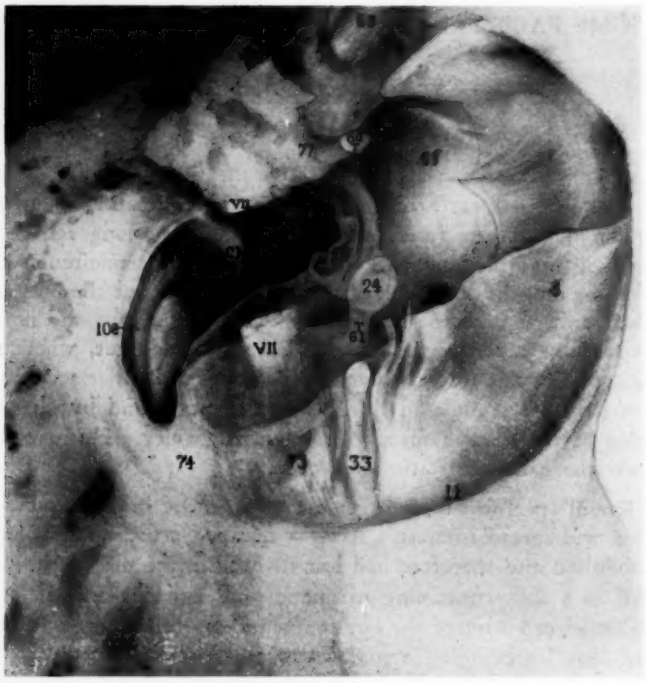



Fig. 1.—Human middle and internal ears seen from above and posteriorly.*

1c. Mastoid air cells; 6. Tympanic membrane; 11. Tympanic annulus; 24. Stirrup: Head (Stapes); 28. Stirrup: Foot-plate; 33. Chorda Tympani; 34. Middle Fossa; VII. Facial Nerve; 59. Pharyngo-tympanic Tube; 60. Tensor Muscle of Tympanic Membrane (60T Tendon); 61. Stapedial Muscle (61T Tendon); 69. Promontory and Tympanic Nerve Plexus; 73. Prominence of Facial Nerve; 74. Prominence of Lateral Semicircular Canal; 77. Cochleariform Process; 87. Niche of Round Window; 106. Vestibule, Membranous: Utricle (106 MN utricular nerve); 108. Vestibule, Membranous: Lateral Semicircular Duct; 112. Lateral Membranous Ampulla (112 CN, cristal nerve).

*Dissected by W. J. McNally, photographed by Wallace Pollock, and photograph retouched by Mrs. Harriet Blackstock Thomson.

## ANATOMY OF THE UTRICLE

The essential structure of an otolith consists of an underlying area of neuroepithelium called the macula, which through its nerve supply, is connected to the nervous system. Overlying the macula is the otolith or otolithic membrane. The utricle, which lies in a horizontal plane, is particularly inaccessible in most animals because of its intimate relationship to the ampullary end organs of the horizontal and anterior vertical semicircular canals. This relationship in man is well shown in Figure 1, which is an actual but retouched photograph of a dissection of the human internal ear. A section of the facial nerve between the markings VII and VII has been excised and it can be seen that the nerve (112 CN) to the crista of the ampulla of the horizontal semicircular canal lies below the level of the facial when seen from the angle at which one carries out the opening of the bony horizontal semicircular canal during the fenestration operation. From this same angle one can see, in the illustration, that the nerve to the utricular macula (MN 106) is at a greater depth still and is actually at a level which corresponds to the top of the stapes footplate (28). Actually the bottom of the utricular macula and its nerve form the roof of the vestibular chamber.

## UTRICULAR FUNCTION

Magnus and deKleijn[3,11] made a detailed analysis of postural reflexes. They distinguished between those of labyrinthine and nonlabyrinthine origin. As the results of experiments on guinea pigs in which they removed the otolithic membranes by the centrifuge method of Wittmaack and from topographical studies made on the labyrinth by Deburlet (see Magnus 1924) they allocated to the utricle, on the one hand, and to the saccule, on the other, certain of the labyrinthine tonic reflexes on the body musculature, certain of the labyrinthine righting reflexes and certain of the labyrinthine eye compensatory reactions.

deKleijn and Lund 1924, reported a set of experiments in which they simultaneously carried out bilateral caloric tests of a guinea pig's ears while the animal was being subjected to tilting stimulation. The animal reacted normally to the tilting. In another group of experiments on the guinea pig, they centrifuged away the otolithic membranes and following this they could still elicit normal nystagmoid movements of the eyes during the caloric stimulation of the semicircular canals but there was no longer any reaction to the tilting. From this, they concluded that the utricles could still respond to gravity stimulation while the semicircular canals are being affected

by caloric stimulation and that, furthermore, the nystagmus from caloric stimulation could still be elicited after the utricles had been inactivated by the elimination of their otolithic membranes. Versteegh,[19] in 1927, succeeded in cutting the nerve to the utricle on one side in a rabbit. These were carefully controlled experiments and he found that following the section of the utricular nerve on the right side the animal's head was turned down to the right. There was a vertical deviation of the eyes, the right eye being down and the left eye up. Vertical compensatory deviation of the eyes became maximum in the left lateral position and was absent in the right lateral position. There was an insignificant diminution of over-all counter-rolling of the eyes during movements about a bitemporal axis. He noted that in the postoperative period there was no spontaneous nystagmus of the eyes and no rolling movements of the animal. He further reported that cold water stimulation of the right ear still elicited a normal nystagmus from the semicircular canals. Versteegh thus showed that all the labyrinthine tonic and righting reflexes described by Magnus and deKleijn were eliminated following section of the utricular nerve.

Tait and McNally[17] reported in 1934 the results of cutting the nerve to the utricle in the frog. This operation could be carried out on one or on both ears. They found it possible also to sever the nerves to all the other labyrinthine end organs and to leave the nerves to the utricles intact. In this way, they were able to experiment with two different types of animal, in one in which only utricles were intact and another in which all the other labyrinthine end organs were intact except the utricles. From this type of experiment these authors concluded that the frog was still able to right itself in the absence of the utricles but if the utricles were removed the frog would not readily carry on sustained forward progression. Such a frog responded with normal compensatory movements to a slow tilt. A frog with only utricles intact, when stimulated by a sudden tipping movement, however, tended to react in a direction opposite to that of true compensation. When such a frog began or ended a movement there was a tendency for pendulation or swaying to take place. These authors concluded that in the normal animal the semicircular canals would, presumably, counteract this pendulation or anticompensatory movement set up by the utricles. An animal with only utricles intact also reacted normally to linear acceleration. When placed on a turntable at some distance from the center of rotation such a frog would react normally to centrifugal force. Such a frog did not react to angular acceleration about a vertical axis when sitting at the center of rotation. The frog from which both utricles had been eliminated

but in which all other labyrinthine end organs were intact could not react normally to centrifugal force. Such a frog when robbed of the two utricles when reacting to angular acceleration was not able to keep its head absolutely level, suggesting that, under normal circimstances, the utricles do serve a purpose even though they are not directly stimulated by rotation about a vertical axis.

Kubo,[9] 1906, Maxwell,[13] 1923, and Ulrich[18] in 1934, all carried out direct stimulation experiments on the utricle in fish. In the experiments of Ulrich the stimulus was delicately applied by using a fine hair. He explored the utricular macula of the pike. He reported that the utricle was sensitive to pressure forward, backward, or to its lateral side, but not to its medial side. He stated that when the applied pressure was restricted to the utricle there was no nystagmus of the eyes. However, if the stimulus effect spreads to the semicircular canals immediately there was nystagmus.

Ross[16] in 1936 recorded electrical responses from individual nerve fibres of the frog's vestibular nerve. He found evidence of a gravity receptor response in the anterior branch of the nerve which he presumed was from the utricle. He also found some gravity responses in the posterior vestibular nerve branch. Ross did not report a resting response from the utricle whereas Lowenstein[10] did report such a resting discharge. In his final analysis, Ross reported that the utricular macula responded to a slow tilt. Ross could not confirm the presence of an anticompensatory response to a quick tilt in the frog's utricle as had been suggested by the experimental results of Tait and McNally in a frog in which only the two utricles remained intact. In 1951, Lowenstein summarized his experiments on the labyrinth in which he recorded electrical responses from individual fibres from the end organs of the vestibular apparatus in the dogfish. He came to the conclusion that the functional ranges of the utricle and saccule overlap. Both contain sense endings which responded to tilting. He also stated that vibrational responses could be recorded from nerve twigs leading from the utricle, the saccule, the macula neglecta and from the lagena. In other words, he found that in the dogfish labyrinth both the utricle and the saccule responded not only to tilting but also to vibrational stimuli.

Fitzgerald and Hallpike[4] analyzed the phenomenon of "nystagmus bereitschaft" which they renamed 'directional preponderance.' Directional preponderance is said to be present to the right when, following caloric stimulation of the labyrinth, there is a tendency for cold caloric stimulation of the left ear and hot caloric stimulation of the right ear to elicit a longer acting nystagmus than does cold

caloric stimulation of the right ear and hot caloric stimulation of the left ear (head back 60 degrees from the vertical plane). Fitzgerald and Hallpike reported having found directional preponderance in certain temporal lobe lesions and in 21 per cent of their cases of Ménière's syndrome. When analyzing this phenomenon in Ménière's syndrome, they came to the conclusion that it was the result of a lesion of the utricle. They stated that a lesion of the left utricle would cause a directional preponderance towards the right. This conclusion was partly based upon the assumption that because the utricle is largely responsible for the tonic reactions from the labyrinth the utricle must play an important part in the production of labyrinthine nystagmus. Jongkees,[7] in 1948, reported finding directional preponderance in about 17 per cent of normal individuals on whom he did alternate hot and cold caloric stimulation of the labyrinth.

Dix and Hallpike,[4] in 1952 described a group of cases under the heading of Positional Nystagmus in which there was vertigo, normal vestibular tests and normal hearing. Positional nystagmus refers to that nystagmus which occurs when the head is placed in certain positions. Dix and Hallpike included the post mortem report of a patient from the above group, in whom a glioma was found to have involved the basal ganglia and the upper part of the brain stem on the left side. There was also histological evidence of degeneration of the spiral ganglia in the cochlea on the right side. In the maculae of the right utricle and the right saccule there was absence of the otolithic membranes, disorganization of the sensory cells and thickening of the subepithelial connective tissue with irregular cellular infiltration. In spite of this widespread pathological finding, the authors concluded that the positional nystagmus in this patient had resulted from disease of the utricular macula.

Jongkees,[7] 1949, reported some experiments which he carried out upon the rabbit, in which he inserted a small metal ball on the macula of the saccule. In two cases out of nine such experiments there was positional nystagmus which he ascribed to damage to the sacular macula. I would point out that in such experiments as this, it is difficult to control and restrict the stimulus so that it is possible that some part of the labyrinth other than the saccule may have been irritated. Because positional nystagmus is associated with certain positions of the head, there has been a tendency, on the part of some clinicians, to assume that the nystagmus probably results from stimulation of the utricle because the utricles are stimulated during a change of head position in relation to gravity. Nylen,[14] in 1950, who has made extensive observations in connection with positional nystagmus

concluded that, whereas positional nystagmus is frequently noted in conjunction with peripheral labyrinthine disease, it is as frequently, or even more frequently, noted in conjunction with a lesion in the vestibular system central to the peripheral labyrinth. Graybiel, Niven and Walsh,[6] in 1952, described experiments in which they submitted humans to stimulation on a large centrifuge. In this apparatus they could, at will, stimulate a person by angular acceleration or by centrifugal force. This was possible because they could maintain the turning at subthreshold levels of angular acceleration while, at the same time, there was being built up a relatively high degree of centrifugal force. Normal individuals, when placed over the axis of rotation, were submitted to pure angular acceleration which is presumed to stimulate the semicircular canals. They reported a feeling of subjective rotation and an apparent rotation of objects in the visual field. These people developed the eye nystagmus that one ordinarily associates with turning stimulation. When the normal patients were seated at some distance from the axis of rotation and were subjected to pure centrifugal force stimulation, which was presumed to stimulate the utricles, they reported that they had experienced an illusion of tilting of the body out of the vertical plane and an apparent displacement of objects in space. Such stimulation did not elicit eye nystagmus. In a group of patients who had suffered from Ménière's syndrome, and upon whom a unilateral labyrinthectomy had been done, were submitted both to pure angular acceleration and to pure centrifugal force stimulation. When asked to compare these induced sensations with their symptoms during a Ménière's syndrome attack it turned out that the symptoms were more nearly reproduced in some patients by angular acceleration (semicircular canal stimulation), in others by centrifugal force (utricular stimulation), and in others the symptoms corresponded to a combination of both types of stimulation. It is important to note that in these experiments of Graybiel and his co-workers pure centrifugal force stimulation, which is presumed to stimulate the utricles, did not elicit eye nystagmus.

## SUMMARY

Experiments have shown that the utricle is stimulated by any change of head position which alters its relation to gravity. It responds to turning which involves centrifugal force and to motion in a straight line—linear acceleration—in either a vertical or horizontal plane. It works in close co-operation with the semicircular canals. The utricle has been shown to exert a dominant influence over labyrinthine tonic reflexes, labyrinthine righting reflexes and labyrinthine compensatory eye deviations.

The best controlled experiments in which stimulation or ablation was strictly confined to the utricle have not elicited eye nystagmus.

Until there is more positive experimental evidence, one views with some doubt, the assumption that such phenomenon as directional preponderance and positional nystagmus result from stimulation or disease of the utricle.

The utricle is a part of the labyrinth which still requires much sound clinical analysis supported by careful experimentation.

1509 SHERBROOKE ST. WEST.

## REFERENCES

1. Breuer, J.: Ueber die Function der Otolithen apparate, Arch. f. d. ges. Physiol. 48:195-306, 1891.

2. deBurlet, H. M.: (See Magnus 1924).

3. deKleijn, A. and Lund, Robert: Über vestibulare Augenreflexe VI. Über die Genese des Kaltwassernystagmus bei Meerschweinchen, Acta Oto-laryng. 6:92-98, 1924.

4. Dix, M. R., and Hallpike, C. S.: The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System, Proc. Roy. Soc. Med. 45: No. 6, 341-354 (June) 1952.

5. Fitzgerald, Gerald, and Hallpike, C. S.: Studies in Human Vestibular Function. 1. Observations on Directional Preponderance ("Nystagmusbereitschaft") of Caloric Nystagmus Resulting from Cerebral Lesions, Brain 65:115-127 (June) 1942.

6. Graybiel, A., Niven, J. I., and Walsh, T. E.: The Differentiation Between Symptoms Referable to the Otolith Organ and Semicircular Canals in Patients with Non-suppurative Labyrinthitis, Laryngoscope 62:924-933 (Sept.) 1952.

7. Jongkees, L. B. W.: (1) Value of the Caloric Test of the Labyrinth, Arch. Otolaryngol. 48:402-417, 1948. (2) Peripheral Nystagmus of Peripheral Origin, Journ. Physiol. 110:3-4:447-449, 1949.

8. Kreidl, A.: Weitere Beiträge zur Physiologie des Ohrlabyrinthes, Sitzungsb. d. k. Akad. d. Wissensch. Math.-Naturw. Cl. 102:149, 1893.

9. Kubo, I.: Ueber die vom N. acusticus ausgelösten Augenbewegungen II. Mitteilung: Versuche an Fischen, Arch. f. d. ges. Physiol. 115:457-482, 1906.

10. Lowenstein, O.: Recording of Responses from Individual End-organs of the Vestibular Apparatus. Proc. Roy. Soc. Med. Section Otol. 133-134 (Dec.) 1951.

11. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

12. Magnus, R., and deKleijn, A.: (see Magnus 1924).

13. Maxwell, S. S.: Labyrinth and Equilibrium, Philadelphia, J. B. Lippincott Company, 1923.

14. Nylén, C. O.: Positional Nystagmus, Jour. Laryngol. and Otol. 64: No. 6, 295-318 (June) 1950.

15. Quix, F. H.: (see Magnus 1924).

16. Ross, D. A.: Electrical Studies on the Frog's Labyrinth, J. Physiol. 86:117-146, 1936.

17. Tait, J., and McNally, W. J.: Some Features of the Action of the Utricular Maculae (and of the Associated Action of the Semicircular Canals) of the Frog. Phil. Tr. Roy. Soc. London, s. B 224:241-286, 1934.

18. Ulrich, H.: Die Funktion des Otolithen, geprüft direkte mechanische Beeinflussung des Utriculusotolithen am lebenden Hecht, Arch. f. d. ges. Physiol. 235: 545-553, 1934.

19. Versteegh, C.: Ergebnisse partieller Labyrinthexstirpation bei Kaninchen, Acta Oto-laryng. 11:393-408, 1927.

20. Aittmaack, K.: (see Magnus 1924).

## XXXVIII

# TRANSPARENT RECONSTRUCTION MODELS

V. E. Negus, F.R.C.S.

London, Eng.

A prolonged study of the anatomy and physiology of the nose and sinuses of animals and man has been divided into three stages.

The first is the collection and dissection of representative species illustrating the organs in fish, amphibia, reptiles, birds and mammals; this collection is assembled in the museum of the Royal College of Surgeons of England, with an offshoot in the Ferens Institute of the Middlesex Hospital.

Stage two has been devoted to observation of the minute anatomy, by means of serial sections taken from various animals and man; the object has been to illustrate the distribution of the different types of mucous membrane, the nerves and mucous glands.

The third stage is directed to physiological problems by experimental investigation, with special regard to olfaction, humidification, warming, ciliary action and respiratory exchanges; it has appeared wise, before attempting to elucidate the various mechanisms, to have a thorough knowledge of the anatomical structure of the organs concerned.

In stage three much work has been accomplished throughout the world, and an extensive literature has been accumulated; but even so certain problems remain to be solved. These concern olfactory acuity and discrimination, the function of the organs of Jacobson, the question of secretion and transudation of moisture, the direction of ciliary streams in animals, and the efficiency of warming of inspired air. It is with stage two that the present communication is concerned.

Investigation of minute detail by the inspection of individual sections with a microscope provides much information, but it is difficult thereby to carry the image in one's mind and to obtain a consecutive picture of the anatomical structure.

The method of wax plate reconstruction allows an enlarged model to be built up, but with various disadvantages. While allowing

---

From the Royal College of Surgeons of England and the Ferens Institute, Middlesex Hospital, London.

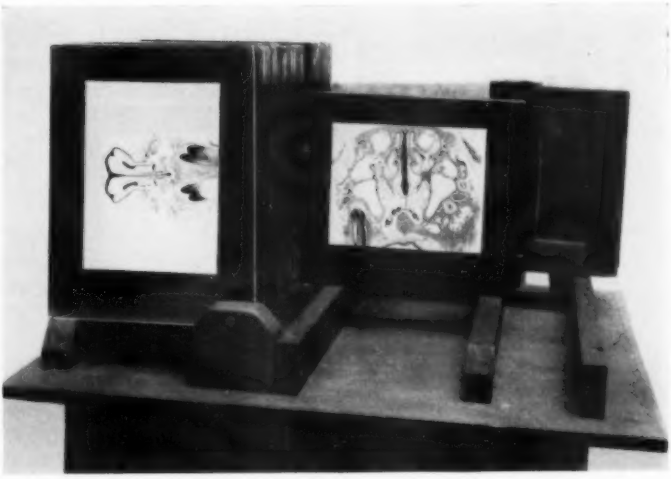


Fig. 1.—Reconstruction of nose of a rabbit (Lepus cuniculus). One section is placed in front of the movable illuminating box. Some of the olfactory ethmo-turbinals are seen and also the large maxillary recess.

external configuration to be shown, it does not give any consecutive view, owing to the opacity of the sheets of wax; it is only possible to divide the model at somewhat wide intervals if the cross sectional view is to be exposed.

There is also the disadvantage that these exposed sections show no more than a tracing of the various structures and do not allow microscopic details to be observed. The method is tedious and, although with many uses, it has not served the purpose required in the present investigation.

The method adopted has been to build up a series of glass plates, each carrying a representation of a cross section and to superimpose these plates in such a way that the transparency can be viewed by placing a light behind it. It is possible to trace by hand enlarged images of each section in various colors and thus to construct a model of a nose or other organ. Such a method has been used by others and in one particular instance has been adopted in the Ferens Institute for the study of the nose of a lizard.

This method of tracing by hand the projection of an enlarged image of a microtome section on to paper, and then transposing it to

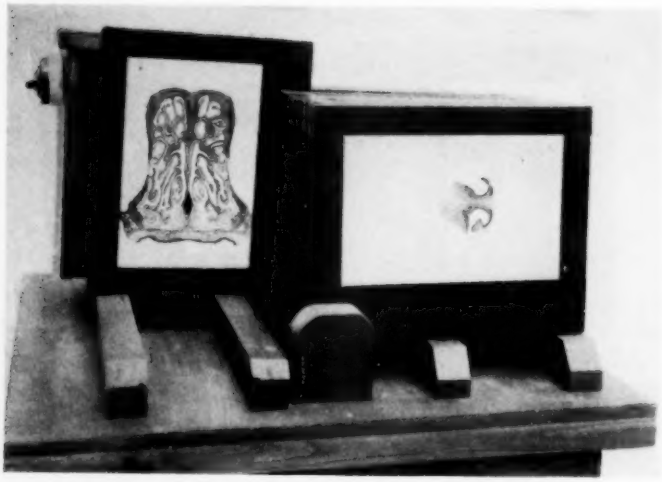


Fig. 2.—Reconstruction of nose of a cat (Felis domestica). The section passes through the ethmoidal region and shows the extensive convoluted turbinals, some extending up into a superadded frontal cavity. The swell body on the lower part of the septum is seen.

glass, has the failing of being tedious and also inaccurate in the representation of minute detail; therefore an alternate method has been adopted.

The animal whose nose and sinuses are to be examined is anesthetized with nembutal, in the case of mammals, and has its tissues fixed intravitally with Wittmaack's solution.

The thorax of the unconscious animal is opened and a cannula is placed in the heart. Blood is washed out by irrigation with Ringer's solution and Wittmaack's solution is then perfused; the composition and quantity used is

| | | |
|---|---|---|
| Potassium bichromate, 5 per cent | | 500 cc |
| Liquid formaldehyde | | 100 cc |
| Glacial Acetic Acid | | 50 cc |
| Aq. dist. | ad | 1000 cc |

The head is removed and is trimmed down to leave the whole of the nasal cavities and sinuses; in the case of the cat and dog this necessitates section behind the posterior limit of the frontal sinus.

After 24 hours in Wittmaack's solution, the specimen, stained yellow by the fixative, is placed in formaldehyde, four per cent in normal saline, for 14 days. It is then transferred to 1 per cent nitric acid for decalcification; the time taken for a frog or toad is about two weeks, but for a cat or dog three weeks is required.

Control of the process of decalcification is attained by radiography, to see that no unaffected bone remains.

The species reconstructed are goldfish (Carassius auratus), newt (Molge vulgaris), toad (Bufo vulgaris), frog (Rana temporaria), lizard (Lacerta Vivipara), slow worm (Anguis fragilis), pigeon (Columba), guinea pig (Cavia porcellus), rabbit (Lepus cuniculus), cat (Felis domestica) and dog (Canis familiaris).

A human foetus of 20 weeks has also been sectioned.

After complete decalcification the specimen is dehydrated in alcohol, graded up to absolute and is then placed in liquid celloidin with stages of 1 per cent up to 12 per cent, two weeks in each; it is finally embedded in 12 per cent celloidin, allowing complete and intact sections to be obtained.

Sections are cut in the coronal (transverse) plane, each being 20μ, or with small specimens 15μ in thickness; the sections are placed on serially numbered sheets of paper.

In the larger specimens every twentieth section is then stained with hematoxylin and eosin and, after being cleared with a special mixed oil, is mounted on a slide. To show particular points of detail it is sometimes necessary to stain and mount every section from a particular part of the block; the same selection is required when dealing with a very short nasal organ, as with a gold fish.

Every twentieth section is then photographed on a quarter plate film (4¼ inch x 3¼ inch), the exposure being sufficiently brief to produce a transparent negative; each negative is aligned with its predecessor as accurately as possible, by focussing onto a fixed part. The next step is fixing the quarter plate negatives on to half plate glass sheets, with accurate alignment obtained by superimposing one film on the next above an illuminated viewing box.

The serial negatives are then enlarged to whole plate size in the case of larger animals, or to half plate size with smaller species. The positive enlargements are purposely underdeveloped, so as to render them transparent, although still showing all the histological details.

The various structures are then tinted, the olfactory epithelium in red, ciliated epithelium in orange, non-ciliated stratified epithelium

Fig. 3.—Serial sections of nose of a slow worm (Anguis fragilis). The illustration gives an idea of the depth of image that can be seen. The nasal fossa is shown, with a projecting concha. Below are seen the paired organs of Jacobson, with the duct opening in the roof of the mouth. (Painting by Miss Hewland, Medical Artist to the Middlesex Hospital.)

—in the nose of the rabbit—in light blue, mucous glands in green and nerves in yellow.

The final step is to mount these tinted enlargements on to sheets of perspex, each 10 inch x 8 inch with whole plates, or 9 inch x 7 inch with half plates; each one is again carefully aligned.

The perspex sheets are provided with a hole at one lower corner, through which a steel rod is passed to carry the whole series. The rod is fixed on to a wooden frame in order that the individual pictures may be rotated to one side or brought in front of a movable box, illuminated with strip lights or bulbs. For a half plate model, two lamps, each of 60 watts, with independent switches, are desirable, and for a whole plate either two 100 watt bulbs or four 60 watt strip lights; the latter provide very brilliant illumination, but with considerable heat. (Figs. 1 and 2)

It is possible to inspect a single slide, so as to observe minute details, or as many as twelve at a time in order to obtain a stereoscopic view and to see into the depths of the structure represented (Fig. 3).

Although such a method may have been used by others, it has not, so far, come to my notice.

A very much better impression can be obtained in this way than by the observation of isolated sections on microscope slides, since there is no necessity of carrying a picture in one's mind.

Not only may a detailed study be carried out regarding the gross and minute anatomy, but furthermore measurements can be made of the relative areas of olfactory and other types of epithelium.

Some of the observations in which these models have been of help have already been published and others will follow in due course.

Many lessons may be learnt from a study of these models. The subdivision of the nose of most mammals into an anterior air conditioning part and a posterior olfactory region (Figs. 1 and 2) becomes obvious; the limited distribution of ciliated epithelium is brought to light and a difficult problem is thereby advanced as to the direction and efficiency of ciliary streams.

The enormous epithelial surface devoted to the warming and moistening of air in macrosmatic animals is illustrated, forcing the conclusion that olfaction is a greater reason for efficiency of air conditioning than is the protection of the lower respiratory tract.

The solution of the function of Jacobson's organs may be facilitated by the ability to examine the structures in all detail. The mode of formation of ethmoidal cells is readily seen by comparing a section of a human fetus with one from a rabbit.

The method described is obviously adapted for the reconstruction of any part of the animal or human body whether normal or pathological.

149 Harley St.

Acknowledgments: Model of the nose of the lizard was constructed by Miss Youngday; intravital fixing of tissues by Mr. J. C. Seymour, Bernard Baron research scholar at the Ferens Institute; processing, cutting, mounting and photographing serial sections by Mr. D. Bishop and assistants; enlarging serial negatives by Mr. Turney of the Photographic Department, Middlesex Hospital; some of the models were assembled in the laboratories of the Royal College of Surgeons by Mr. Bartlett; measurements of epithelia by Mr. Jamieson, student in the Medical School of the Middlesex Hospital.

REFERENCES

1. Negus, V. E.: Observations on the Comparative Anatomy of the Nose. Acta Otolaryngologica 44:13, 1954.

2. Negus, V. E.: Proceedings Anatomical Society of Great Britain 88:558, 1954.

3. Negus, V. E.: Introduction to the Comparative Anatomy and Physiology of the Nose. Annals Royal College of Surgeons of England 15:141, 1954.

# XXXIX

# THE ROLE OF THE CEREBRUM IN HEARING

JOHN E. BORDLEY, M.D.

AND

HARRIET L. HASKINS, A.M.

BALTIMORE, MD.

The biologic mechanism by which physical sound is perceived extends from the external ear to the cortex of the brain. For the present discussion, lesions of the outer and of the middle ear have no interest. They damp the intensity of the sound that reaches the inner ear but do not otherwise affect tonal combinations. A simple increase in intensity therefore suffices to overcome the effect of any such lesion on the perception of sound. Lesions of the portion of the hearing mechanism that extends from the neuro-epithelium of the cochlea to the cerebral cortex present a more complex, and frequently a baffling, problem to the otologist.

Among the patients whose records are to be considered today are some who, paradoxical as this statement may seem, can hear but who are deaf. They have the physiological ability to hear but are unable to analyze and to comprehend what they hear. Sounds reaching them have no meaning. To the clinician, and certainly to the patient, this is a form of deafness.

The child born with such a handicap fails to learn the rudiments of communication. He cannot understand what he hears, and because sound must have some meaning before speech is learned he does not acquire language. When such a handicap develops even to a moderate degree in the later decades of life the afflicted individual suffers deafness. This deafness is characterized by inability to comprehend conversation. unless that conversation is delivered under the optimum circumstances of quiet, slow pace, and concentration. Group conversation causes a severe breakdown in ability to keep up with the subject. Such people complain that they hear, but that people fail to speak

---

From the Department of Otolaryngology of the Johns Hopkins University School of Medicine and of the Johns Hopkins Hospital.

Paper presented in a Symposium on Sound and Its Perception, at the Ninety-Second Annual Meeting of the National Academy of Sciences, held in Washington, D. C., April 25, 1955.

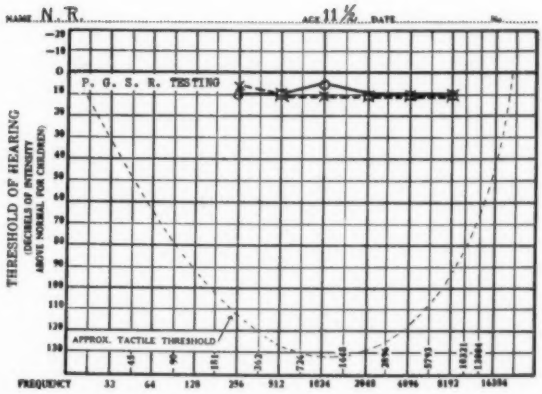



Fig. 1.—The objective audiograms, obtained by testing with the psychogalvanic skin-resistance method, of a child who at 11 years of age has not learned to speak. By standard (subjective) audiometry thresholds of hearing cannot be determined because sounds have no meaning to this child with a defective brain.

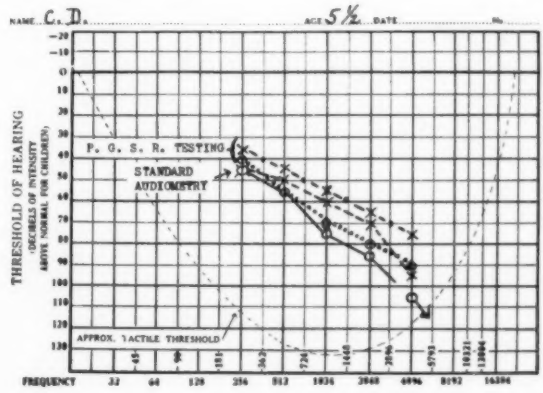



Fig. 2.—The subjective audiograms (Standard Audiometry) and the objective audiograms (P. G. S. R. Testing) of a child whose brain functions normally.

clearly to them. Disability in this group is far greater than that seen in their contemporaries with similar thresholds for hearing by standard tests. In this group, the hearing aid adds only to their confusion and they cannot wear it successfully. As Pauls[1] has pointed out, such disability seems to stem from a fundamental breakdown in auditory discrimination of the elements and meaning of speech.

An extreme example is this child, aged 11, who was born by a difficult breech presentation 48 hours after the mother's membranes ruptured. He was cyanotic at birth and for some time after. He had several convulsive seizures. He first held up his head at 18 months. He sat up at two years. He walked at three and one-half years. His gait is still wide and unsteady. At 11 he is partially toilet trained, and feeds himself only occasionally, and then with his bare hands. He has no speech. He will occasionally turn his eyes toward some new sound. He makes no attempt to communicate. Psychometric evaluation places him at the idiot level. By objective audiometry his hearing is normal (Fig. 1). Sound means nothing to him. He hears, but he is deaf.

In contrast to the preceding child are the records of this little girl now aged eight years. She was born following toxemia of pregnancy. At 18 months she paid no attention to sound. She had developed no speech, but was quite vocal in her play. She was subject to temper tantrums and was anti-social, being ill-tempered and overbearing. By objective audiometry her hearing proved to be severely impaired. She was given a hearing aid at 18 months, and started on rehabilitative training. Today she stands in the top third of her class in a regular school. She is with her chronological equals. Her speech is not perfect, but her ability to communicate is satisfactory. She is deaf, but she can hear.

A review of her hearing tests reveals that when she became five and one-half years old and could be studied by standard subjective techniques, her thresholds were substantially in agreement with repeated earlier objective audiograms (Fig. 2).

The objective technique for audiometry employed in the Hearing and Speech Center at the Johns Hopkins Hospital, known as psychogalvanic skin-resistance audiometry, is based on the Pavlovian conditioned reflex.[2] A pure tone from a standard audiometer is used as a warning signal about three seconds before a mild but uncomfortable electric shock to the calf of the leg. The shock results in increased activity of the sweat glands. This lowers the resistance of the skin. Repetition of the sound-shock sequence will after eight or ten tries, produce conditioning whereby the subject begins to sweat when the

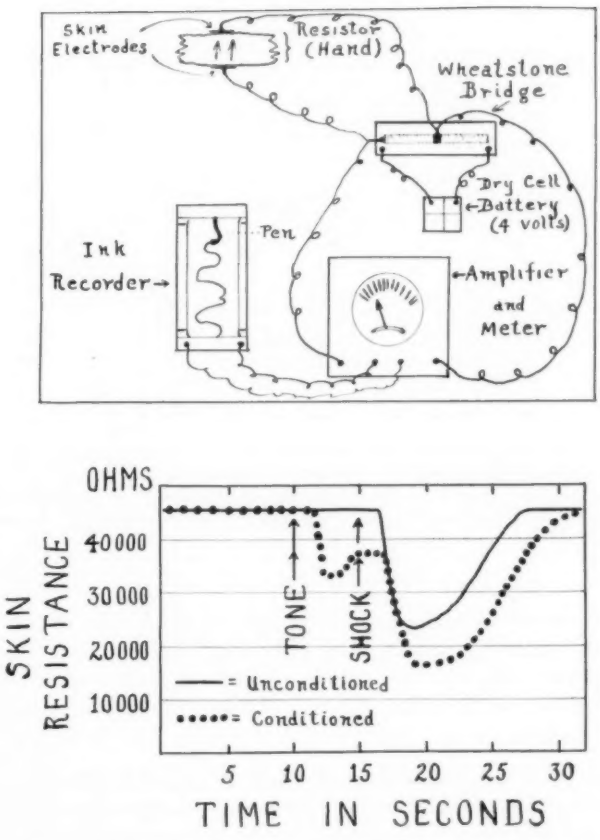



Fig. 3.—These two sketches show schematically the psycho-galvanic method of testing hearing. The changes in resistance of the skin to the passage of a minute electric current can be read from the meter and are permanently recorded, by an "ink-writer," on a moving strip of paper.

Fig. 4.—An example of the type of "ink-writer" record obtained by the psycho-galvanic skin-resistance method of determining the threshold of response to a pure tone from an audiometer, after the patient is conditioned. (From Bordley and Hardy[5]) The thresholds thus recorded are later, for convenience of reference, charted on standard audiogram forms.

tone is sounded in anticipation of the coming shock. In other words, instead of pressing a button when the tone rises above threshold, the patient sweats (Fig. 3). By raising and lowering intensities, thresholds for each tone can be obtained (Fig. 4) and plotted on a standard audiogram form.

This skin-resistance change was first described by Feré in 1888.[3] The method for graphically recording such changes was devised by Richter in the research laboratories of the Department of Psychiatry at the Johns Hopkins Hospital in 1928.[4]

That the end organ for such a reflex lies within the ear and that the reflex is not the result of some tactile stimulus is shown by these records on a Rhesus monkey (Fig. 5). After initial psychogalvanic skin-resistance audiograms were made and repeated on different days, without significant changes in thresholds, the auditory nerve was completely sectioned. Following full physical recovery, repeat audiograms showed the hearing on the unoperated side had not changed. When masking was used on the normal ear it was found impossible to get

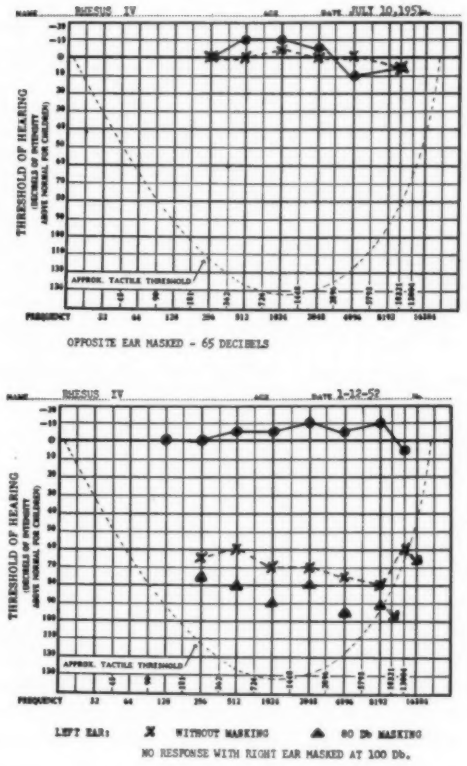



Fig. 5.

skin-resistance responses to intense tonal stimulation of the operated ear.

Correlation between standard and psychogalvanic skin-resistance audiometry in adults is quite satisfactory.[5] This has been shown by hundreds of comparisons, using both individuals with normal hearing and those with well documented auditory lesions.

The question of where such a conditioned reflex is mediated in the brain is still unsettled. Certain facts have already been demonstrated, however, that seem to be significant. Wang, working in Richter's laboratory, has recently shown, in some as yet unpublished experiments, that the shock-sweat sequence can be abolished in cats after

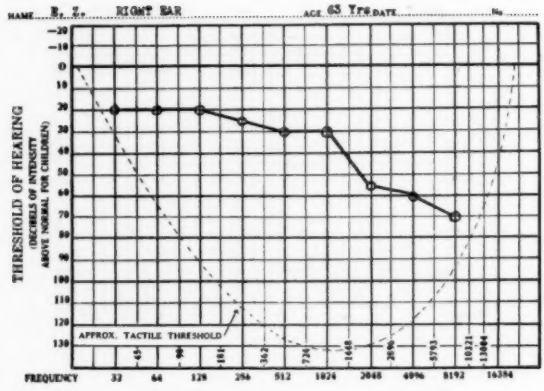



Fig. 6.—The subjective audiogram, by standard audiometry, of an ear in which histologic examination (Crowe, Guild and Polvogt[9]) revealed a partial atrophy of the cochlear nerve sufficient to explain the hearing impairment.

transection of the brain on the plane between the colliculi and the rostral border of the pons. Bromiley[6] reported in 1948 that he had been able to develop a conditioned response in a decorticate dog; that the dog could discriminate between light and sound. Wang and Nager,[7] of the Otological Laboratory of the Johns Hopkins University, recently successfully conditioned a decorticate cat to a buzzer and to a bell by the psychogalvanic method. Pavlov[5] in 1912 stated that he thought the site of such conditioning must lie within the cerebrum.

In 1934 Crowe, Guild and Polvogt[9] reported observations on 79 temporal bones which had been serially sectioned. The hearing levels of these ears had been evaluated by standard audiometry and tuning fork tests before death. These temporal bones were divided according to microscopic findings into three groups. The first group had atrophy of the organ of Corti and associated degeneration of some of the nerve fibers in the lower basal turn of the cochlea. Their hearing exhibited a sharp drop in acuity for the high tones. The second group had partial atrophy of nerve fibers in the osseous spiral lamina with some loss of spiral ganglion cells in the lower basal turn of the cochlea. This group exhibited a gradual type hearing loss from low to high tones (Fig. 6). The third group of 17 ears had no lesions that could explain the hearing loss. The audiograms of this group were similar to those of the second group.

Fieandt and Saxen,[10] in 1937, reporting on the temporal bone findings in 33 ears with demonstrated nerve type deafness also had three general groupings. Their third group contained one case. This was a man age 79, with no demonstrable pathology in the temporal bone.

Crowe and his associates in 1934 and Fieandt and Saxen in 1937, speculated that the explanation for the impairment of hearing in those cases with no temporal bone pathology might be found in the central nervous system. Schuknecht[11] in a very recent paper, stated that the degenerative changes in the cochlea might well parallel those found in the aging brain. He felt that loss of the second, third and fourth order neurons might play a part in old-age deafness.

In 1953, one of the authors (H. H.), working with a group of older adults in the Hearing and Speech Center at the Johns Hopkins Hospital, found that a number of these individuals could not be fitted satisfactorily with a hearing aid. Similar observations have been made in almost all audiologic centers. By standard audiometry these individuals had similar thresholds to many other patients who had been successfully fitted with hearing aids. In trying to find some cause for this failure to accommodate to amplification, a number of these patients were tested by psychogalvanic audiometry. In each case the psychogalvanic thresholds were significantly better than those by standard audiometry. In fact, the hearing levels established by this technique were so near normal thresholds that it was easy to see why amplification could not be tolerated.

A group of these patients were selected for further study. The criteria for selection were a suitable standard audiogram for amplification but inability to wear a hearing aid.

Significant observations common to this group are:

1. The objective audiogram (PGSR) is significantly better than the standard subjective audiogram.

2. By speech audiometry, thresholds in decibels for awareness of sound and those for understandability of speech are much further separated than in persons using amplification successfully.

3. Ability to understand simple words when spoken slowly is good. As the speed of delivery is increased there is abnormally increased difficulty in the understanding. That is, in the test situation where words are presented very slowly with a "breather" given by the tester between each word, such as the simple sentence, "That is right," the discrimination score is much better than when similar test words

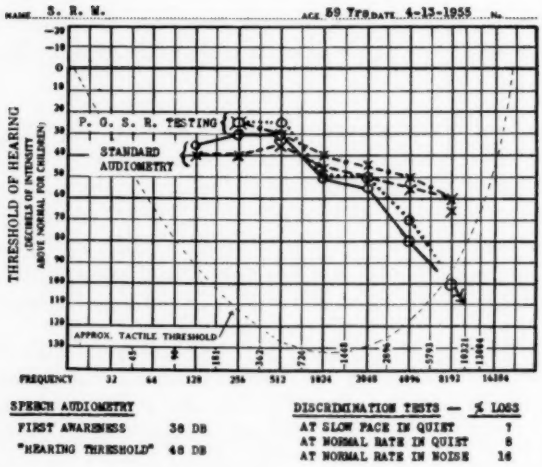

NAME S. R. M.
AGE 59 YRS DATE 4-13-1955
P. G. S. R. TESTING
STANDARD AUDIOMETRY
APPROX. TACTILE THRESHOLD
THRESHOLD OF HEARING (DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384
SPEECH AUDIOMETRY
FIRST AWARENESS 30 DB
"HEARING THRESHOLD" 40 DB
DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 1
AT NORMAL RATE IN QUIET 8
AT NORMAL RATE IN NOISE 16

Fig. 7.—Records of the indicated tests of a patient with "nerve deafness" who successfully uses a hearing aid. See text and compare with the data shown in Figure 8.

are given at a normal rate. If tape recorded sequences of the same words are given without the possibility of a pause the discrimination score is very poor. They all do better in quiet than in noise. Any increase in the complexity of the test situation such as using the Békésy self recording technique for pure tone audiometry cause a further depression of apparent thresholds.

The records of an individual who successfully wears a hearing aid will highlight the points of difference under discussion. This man has similar subjective and objective pure tone thresholds (Fig. 7). In speech audiometry he has only the average normal spread of about ten decibels between awareness of sound and understanding of speech. The word discrimination is good at normal speed.

The data for four of the patients who cannot wear hearing aids are presented in Figures 8 and 9. All of these individuals are past middle age; all of them have been very successful in their chosen field of work in life. None has had any previous ear difficulty, or difficulty with hearing. The onset of their present complaint dates within the past three or four years. They feel they have developed a gradual loss of ability to hear normally. Their chief complain is that people do not speak intelligibly to them any more; that they must face a person and

talk only to him as an individual in order to understand. They have great difficulty communicating in noisy places. Group conversations, such as committee meetings, have become almost impossible. If people raise their voices to help them, it adds to their confusion.

In each case the standard subjective audiogram has been repeated many times and has not varied significantly. Such audiograms present a favorable level for the use of amplification. None of these individuals have been successfully fitted with a hearing aid. In each instance the thresholds by psychogalvanic skin-resistance audiometry have been much better than those obtained by standard audiometry. Awareness of sound is about 20 decibels better than speech understanding instead of the usual 6 to 10 decibels. Discrimination scores are poor at normal pace.

Certain animal experiments would seem to help explain such a peculiar form of deafness. In 1911 Babkin,[12] a pupil of Pavlov, reported tests made on a dog that lived three years after removal of the posterior portion of both cerebral hemispheres. The dog could be conditioned to simple sounds, but he could never relearn the more complicated sequences of sounds, even his own polysyllabic name, to which he could be conditioned before the operation. Babkin felt that the dog's ability to discriminate tonal patterns was severely impaired. In discussing this and other experiments in his laboratory Pavlov stated in 1912,[8] "From the standpoint of the conditioned reflexes the cerebral hemispheres appear as a complex of analyzers, whose functions are: to decompose the intricacy of the outer and inner worlds into their separate elements and components, and further, to connect all of these with the manifold activity of the organism." In 1952 Meyer and Woolsey[13] noted that after extensive bilateral ablations of the auditory cortex, some animals could acquire simple conditioned response to a sound but could not discriminate between frequencies. In 1953, Diamond and Neff[14] reported that after complete bilateral ablation of the auditory cortical areas in a cat, as determined by the evoked potential method, the discriminatory habit was lost and could not be relearned.

It would seem, then, that in some instances the conditioned reflex type testing as employed in psychogalvanic skin-resistance audiometry measures only the efficiency of the auditory end organ and the subcortical auditory pathways, and that measurements by subjective audiometry represent the performance of the hearing mechanism as a whole from the external ear through the auditory cortex. If such is the case it would seem reasonable to think that those individuals with good hearing by objective audiometry but poor hearing by subjective audiometry, with poor discrimination performance, and inability to use

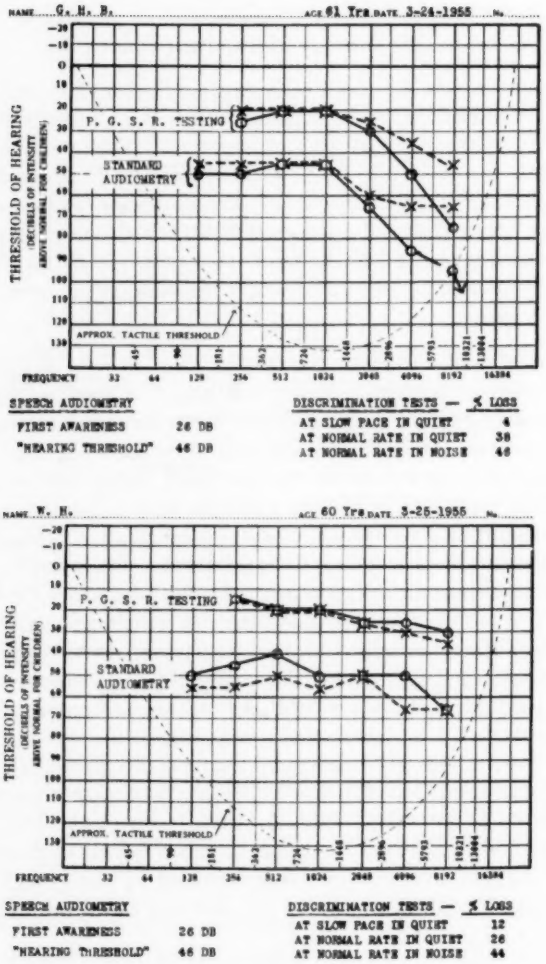

NAME G. H. B. AGE 61 Yrs. DATE 3-24-1955 No.
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
P. G. S. R. TESTING
STANDARD AUDIOMETRY
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384
SPEECH AUDIOMETRY
FIRST AWARENESS 26 DB
"HEARING THRESHOLD" 46 DB
DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 4
AT NORMAL RATE IN QUIET 38
AT NORMAL RATE IN NOISE 48
NAME W. H. AGE 60 Yrs. DATE 3-25-1955 No.
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
P. G. S. R. TESTING
STANDARD AUDIOMETRY
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384
SPEECH AUDIOMETRY
FIRST AWARENESS 26 DB
"HEARING THRESHOLD" 46 DB
DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 12
AT NORMAL RATE IN QUIET 26
AT NORMAL RATE IN NOISE 44

Fig. 8.—Records of the indicated tests of intelligent, co-operative patients who get no satisfaction from trying to use a hearing aid.

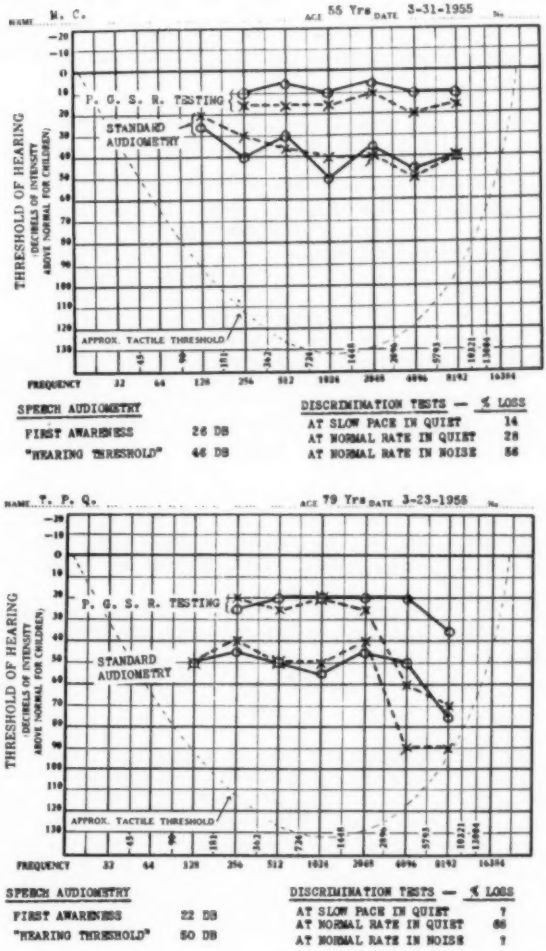

NAME M. C.
AGE 55 Yrs DATE 3-31-1955
P. G. S. R. TESTING
STANDARD AUDIOMETRY
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384
SPEECH AUDIOMETRY
FIRST AWARENESS 26 DB
"HEARING THRESHOLD" 46 DB
DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 14
AT NORMAL RATE IN QUIET 28
AT NORMAL RATE IN NOISE 56
NAME T. P. Q.
AGE 79 Yrs DATE 3-23-1955
P. G. S. R. TESTING
STANDARD AUDIOMETRY
THRESHOLD OF HEARING
(DECIBELS OF INTENSITY ABOVE NORMAL FOR CHILDREN)
APPROX. TACTILE THRESHOLD
FREQUENCY 32 64 128 256 512 1024 2048 4096 8192 16384
SPEECH AUDIOMETRY
FIRST AWARENESS 22 DB
"HEARING THRESHOLD" 50 DB
DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 7
AT NORMAL RATE IN QUIET 86
AT NORMAL RATE IN NOISE 7

Fig. 9.—Records of the indicated tests of two further patients in the category of those of Figure 8.

amplification suffer fundamentally from damage to the cerebrum, probably to the cerebral cortex. The aging process is known to cause deterioration of the different organs of the body at a different rate in each individual. Many individuals may, of course, have involvement not only of the cerebral hearing mechanism but also of the peripheral hearing mechanism.

The clinical and laboratory observations, considered together, suggest that the difference between the hearing thresholds by subjective and by objective audiometry indicates approximately the degree of cerebral sclerosis of the auditory area.

JOHNS HOPKINS HOSPITAL.

## REFERENCES

1. Pauls, M. D.: Phonemic Discrimination in Relation to Difference Limens for Intensity in the Perceptively Deafened Ear. Summaries of Doctoral Dissertations, Northwestern University, 17:113-117, 1949.

2. Bordley, J. E., Hardy, W. G., and Richter, C. P.: Audiometry with the Use of Galvanic Skin-Resistance Response. Bull. Johns Hopkins Hosp. 82:569 (May) 1948.

3. Feré, C.: Note sur les modifications de la résistance électrique sous l'influences des excitations sensorielles et des émotions. C. R. Soc. Biol. Paris, 5:217, 1888.

4. Richter, C. P.: Electrical Skin Resistance; Diurnal and Daily Variations in Psychopathic and in Normal Persons. Arch. Neurol. and Psychiat. 19:488-508 (Mar.) 1928.

5. Bordley, J. E., and Hardy, W. G.: A Study in Objective Audiometry with the Use of a Psychogalvanometric Response. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 58:751-759 (Sept.) 1949.

6. Bromiley, R. G.: Conditioned Responses in a Dog after Removal of Neocortex. Jour. Comp. and Physiol. Psychol. 41:102-110 (Apr.) 1948.

7. Wang, G. H., and Nager, G. T.: Personal Communication.

8. Pavlov, I. P.: Summary of Results of Removal of Different Parts of the Cerebral Hemispheres. (A lecture published in the Transactions of the Society of Russian Physicians, 1912-1913: and as Chapter 18 of Pavlov's Lectures on Conditioned Reflexes, 1:193-204, translated by W. H. Gantt.) International Publishers Co., Inc., New York, 1928.

9. Crowe, S. J., Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High-Tone Deafness. Bull. Johns Hopkins Hosp. 54:315-379 (May) 1934.

10. Fieandt, H. von, and Saxen, A.: Pathologie and Klinik der Altersschwerhörigkeit. Acta Oto-laryng., Suppl. 23, 1-102, 1937.

11. Schuknecht, H. F.: Presbycusis. Paper read at Meeting of the Amer. Laryngol., Rhinolog. and Otolog. Soc., Hollywood, Florida, March 17, 1955. In Press.

12. Babkin, B. P.: Further Studies Upon the Normal and the Injured Acoustic Analyser in the Dog. Proc. Russian Med. Soc. in St. Petersburg, Vol. 78, 1911. (Cited from Pavlov, Ref. 8.)

13. Meyer, D. R., and Woolsey, C. N.: Effects of Localized Cortical Destruction on Auditory Discriminative Conditioning in Cat. Jour. Neurophysiol. 15:149-162 (Mar.) 1952.

14. Diamond, I. T., and Neff, W. D.: Role of Auditory Cortex in Discrimination of Tonal Patterns. Fed. Proc. 12:33 (Mar.) 1953.

XL

# THE MAXIMUM STRENGTH OF THE TYMPANIC MUSCLES

ERNEST GLEN WEVER, PH.D.

JACK A. VERNON, PH.D.

AND

MERLE LAWRENCE, PH.D.

PRINCETON, N. J.

It is well established, mainly on the basis of cochlear potential studies on cats and guinea pigs, that the primary function of the tensor tympani and stapedius muscles is a protection of the inner ear against overstimulation by sounds. When the stimulation is excessive there is a reflex excitation usually of both these muscles, and their contractions bring about a modification of the transmission characteristics of the ossicular mechanism. Typically this modification consists of a reduction in transmission, to an extent that varies with the degree of muscular tension developed.

Only limited observations have been made on the maximum tensions of which these muscles are capable. Lorente de Nó[1] reported a measurement of this tension as 1.2 grams for the tensor tympani muscle of the rabbit, and Wever and Lawrence[2] obtained evidence that led to an estimated maximum of about one gram for the stapedius muscle of the cat. The present experiments seek to extend our knowledge of the strengths of these muscles in the cat.

## METHOD AND PROCEDURE

The animals were anesthetized with sodium pentobarbital and then decerebrated. The middle ear structures were exposed by a wide opening of the auditory bulla and removal of its bony septum. The spicule of cartilage that serves as a mooring post for the chorda tympani nerve was removed also, along with the nerve itself. The view of the structures thereby obtained was like that exhibited in Figure 1.

A fine silk thread was tied to the tendon of one or the other of the muscles and connected to a strain gauge. In order to obtain

This research was supported by the Office of Naval Research under Contract N6-onr-270 (3) and by Higgins funds allotted to Princeton University. Acknowledgement is made of the assistance of Walter E. Rahm, Jr., in connection with the strain gauge circuit.

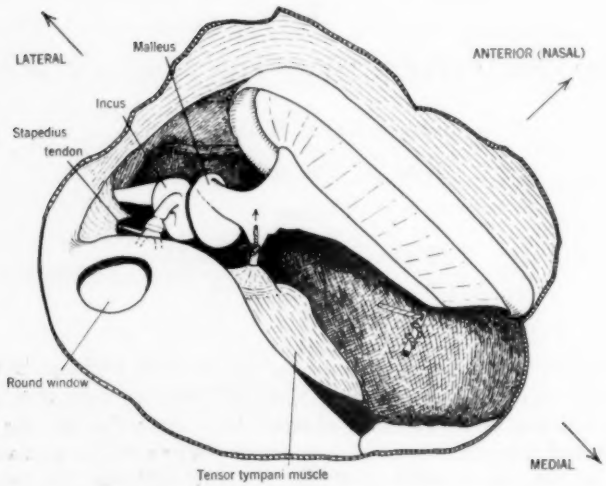



Fig. 1.—A ventromedial view of the middle ear of the cat, right side. A thread is shown tied about the bony spicule of the malleus to which the tensor tympani muscle is attached. This thread was led out of the bulla cavity, in a direction indicated by the arrow, to the operating arm of a strain gauge. For measurements on the stapedius muscle, another thread was tied about the stapedius tendon and led across the bulla in a medial direction to the end of a needle to which it was secured; only a portion of this thread and its connection to the end of the needle are shown in the drawing.

the proper direction of pull the arrangement varied a little for the two muscles. For the tensor tympani muscle the thread was led out of the bulla in a ventrolateral direction to the operating arm of the strain gauge. The position of this thread and its direction of action are indicated in the figure. For the stapedius muscle it was necessary to add an extension to the operating arm of the strain gauge so that its end could be inserted into the medial part of the bulla cavity. This end was a slender steel needle with an eye at its tip; the thread, which had already been tied to the stapedius tendon, was passed through the eye, drawn taut as the extension was lowered into place, and finally secured about the needle's shank. The tip of this needle is represented in the figure; the thread ran from its attachment to the stapedial tendon to the position as shown.

For both muscles, our effort was to run the thread in the direction of the tendon, but in most of the cats we found ourselves restricted by the conformation and size of the bulla, and for the stapedius muscle in particular we often had to be content with a direc-

tion some 15 to 20 degrees away from the estimated normal. This variation does not produce any serious error, however. The error is equal to 1 minus the cosine of the angle of departure from the normal direction, and it will not exceed 10 per cent for any deviation up to 25 degrees.

The strain gauge consisted of four resistance wires in a Wheatstone bridge arrangement. As used here, the bridge was actuated by a sinusoidal current of 400 cycles at 10 volts. The bridge was balanced under zero tension by varying the center tap position on a shunt resistance. The application of tension produced a stretching of two of the resistance wires and a relaxation of the other two, thereby altering the resistance ratios and unbalancing the bridge. This unbalance produced bridge currents that were read on a sensitive vacuum tube voltmeter. The system was calibrated for every condition of use by suspending weights from the operating arm. The first measurements were made with a gauge whose maximum range was 30 grams, and later measurements with one whose range was 10 grams. The latter instrument was sensitive to 3 milligrams as used for the tensor tympani measurements and to 1.3 milligrams when the extension arm was added as in the stapedius measurements.

The measurements were carried out under two conditions: (1) when the middle ear was intact and its structures remained undisturbed except for the opening of the bulla and the attachment of a thread as described, and (2) after freeing the muscles from the ossicular chain. In this freeing operation a small piece of bone was left attached to the tendon to aid in the securing of the thread. For the tensor this piece was the bony spicule of the malleus to which the tendon is attached, and for the stapedius it was the head and neck of the stapes or sometimes this whole ossicle.

The muscles were stimulated electrically with sinusoidal currents usually at a frequency of 1000 cycles, by means of two needle electrodes located in the tissues of the head so as to give a field running lengthwise through the muscle. The placement of both electrodes thus was different for the two muscles. For usual stimuli only the muscle lying between the two electrodes was made to contract, but both muscles could be excited by raising the current strength to high levels.

The usual procedure for making strain measurements was first to balance the bridge when the strain was zero, then to retract the gauge (by means of a micromanipulator) so as to pull the thread straight and put a slight tension on it. This initial tension was normally 0.6 grams for the tensor tympani muscle and 0.3 grams

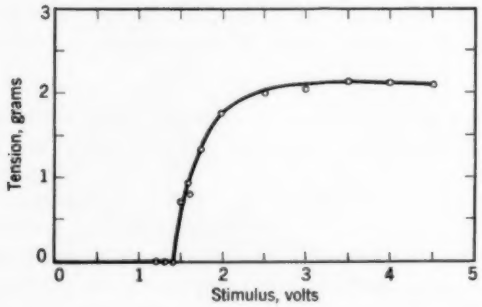



Fig. 2.—Strain function for the tensor tympani muscle, under electrical stimulation.

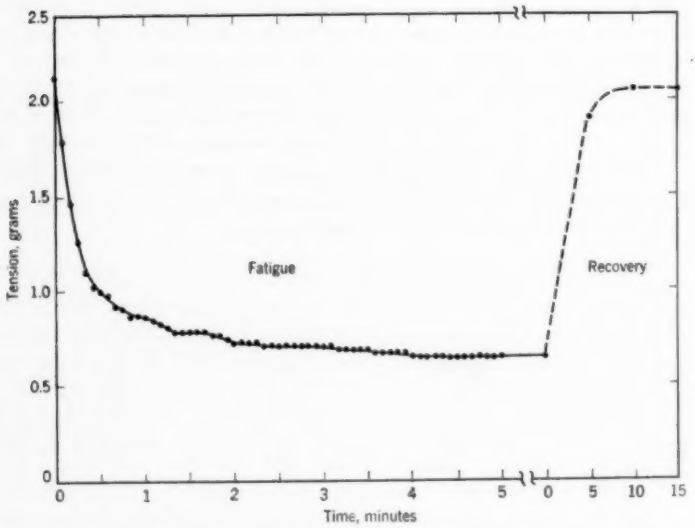



Fig. 3.—Fatigue and recovery functions for the tensor tympani muscle.

for the stapedius muscle. Then the muscle was stimulated, usually at intensities that varied from zero upward by small steps of 0.1 or 0.2 volts until the threshold of contraction was reached, and thereafter by somewhat larger steps (usually 0.5 volts) until a maximum contraction was obtained. Ordinarily a rest interval of 30 seconds or more followed every stimulation. The action was also observed through a binocular microscope at 15 times magnification.

## RESULTS

A typical strain function for the tensor tympani muscle, when the middle ear mechanism was intact, is shown in Figure 2. Here, as will be seen, the threshold was passed at 1.4 volts and the maximum contraction was elicited at 3.5 volts. It should be said, however, that these stimulus values have no absolute significance, as they depend both upon the internal impedance of the generator (which was 600 ohms) and upon the amount and kinds of tissue lying between the electrodes. As would be expected for an increasing stimulation of this kind, the contraction of the muscle rises rapidly at first and then more slowly until a maximum is attained. In this instance the response remained constant at 2.1 grams for still larger stimulations, but we have often observed a true maximum, a point beyond which the response falls off appreciably as the stimulation is increased.

Results of this kind were obtained on nine ears. Usually it was possible, within the time available, to make the measurements only on one of the muscles, though in three instances they were carried out on both. Table 1 shows the maximum tensions obtained under the two conditions already mentioned: under the restraints afforded by the normal connections to the ossicular chain and after these restraints had been removed. As will be seen, there are large differences among the ears, but certain tentative conclusions can be drawn. In general the tensor tympani muscle is considerably stronger than the stapedius, as would be expected from its larger size. For the tensor tympani muscle, the "free" condition gives larger tensions than the "restrained" condition in three out of five instances, and in the other two the observed maximums are practically the same. In the single comparison that the data afford for the stapedius muscle there is no significant difference for the two conditions. The mean values of the maximums are 3.5 grams for the tensor tympani muscle in the "restrained" condition and 4.4 grams in the "free" condition, and for the stapedius muscle the means are 1.6 grams for both conditions. These means are not changed appreciably if we use all of the series for every animal rather than the maximum value obtained for each, for though there were variations from series to series for a given ear, these variations were small in comparison with those for different ears. Thus the

TABLE 1.

| CAT NO. | MAXIMUM TENSION IN GRAMS | | | |
|---|---|---|---|---|
| | TENSOR TYMPANI MUSCLE | | STAPEDIUS MUSCLE | |
| | "RESTRAINED" | "FREE" | "RESTRAINED" | "FREE" |
| 1021 R | 2.1 | .... | .... | .... |
| 1022 R | 1.6 | 2.9 | .... | .... |
| 1023 R | 0.8 | 2.6 | .... | .... |
| 1027 R | .... | .... | .... | 1.2 |
| 1074 R | 5.7 | 5.4 | 3.0 | 2.8 |
| 1080 R | 1.6 | 5.3 | 0.3 | .... |
| 1081 R | .... | .... | .... | 0.7 |
| 1084 R | 6.6 | 6.0 | 2.0 | .... |
| 1087 R | 5.9 | .... | 1.0 | .... |
| means | 3.5 | 4.4 | 1.6 | 1.6 |

differences shown represent actual differences in strength among the muscles studied.

As already indicated, we attempted to run the thread to the strain gauge along the line of the tendon. For the stapedius muscle this is clearly the line of action, for its tendon passes through a narrow bony channel on its way to the attachment to the stapes. The tendon of the tensor tympani muscle, however, is not guided in this fashion, and its free portion is very short. Hence there is a possibility for tension to be exerted in various directions within a solid angle of about 90 degrees. In some of the experiments the direction of the thread leading from the tensor tympani muscle was systematically varied, within the limits imposed by the anatomical situation, to test this possibility. For small variations about the estimated normal there was no significant alteration of the tension, as would be expected. When, however, the thread was run in a direction that was obviously different from the normal the effective tension was markedly diminished. In one experiment the thread from the tensor was run in a medial direction, the same direction as used in the measurements on the stapedius muscle. This was accomplished by use of the extension arm, whose end was placed just as indicated in Figure 1, but with the thread extended medially from the tensor tendon. The measured tension for maximum contraction was 0.6 grams—about one-tenth the tension exerted by the same muscle when meas-

ured in the usual ventrolateral direction. It should be noted that this medial tension, as exerted upon the ossicular chain, is just contrary in direction to that exerted by the stapedius muscle. The two muscles therefore are partly counteractive in their tensions upon the ossicular chain.

Visual observations were made along with the strain gauge measurements in order to obtain further particulars of the action. The stapedius muscle itself was not seen, as it remained buried in its cavity in the temporal bone, but the movements of its tendon and of the stapes were observed. The tensor tympani muscle was in clear view (as Figure 1 has shown), for its proximal portion lies in a recess in the superior part of the bulla and its distal portion extends into the bulla cavity. Therefore its movements along with those of the manubrium of the malleus could be noted in detail. Often we found that for weak stimuli—those in the region of the threshold—a slight twitch of the medial portion of the tensor tympani muscle, evidently involving only a few of its fibers, could be seen when there was no obvious movement of the manubrium and no alteration of tension was registered by the strain gauge. As the stimulation was increased the responses became more vigorous and seemed to involve the whole muscle. Then the visible movements and the strain gauge measurements followed a parallel course.

In nearly all the trials, including those whose results are given in Table 1, a standard initial tension was applied to the muscle, as already mentioned, but in some of the experiments this initial tension was varied, for a constant intensity of stimulation, to discover the effect upon the muscle action. The most extensive measurements were made on the tensor tympani in the "free" condition. In one series the initial tension was varied in different trials from 0.6 to 3.1 grams, and though the total tension increased with the initial loading, the net tension was essentially unchanged, varying only randomly between 5.2 and 5.4 grams. Similar results were obtained for the stapedius muscle when "restrained," except that smaller tensions were recorded when the initial loading was below 0.2 grams, possibly because then the forces developed by the muscle were no longer communicated properly to the gauge but were dissipated in part in taking the kinks out of the thread.

For the small and moderate degrees of stimulation the tension was maintained for the period of the stimulation, which in normal trials did not exceed one second. For the larger stimulations this was no longer the case; the tension promptly attained its maximum and then fell away to perhaps a third of this value. When a moderate intensity of stimulation was maintained for long periods there was

a gradual diminution of the tension. Figure 3 shows results of an experiment in which the stimulation of the tensor tympani muscle was continuous for five minutes, during which the tension was noted at frequent intervals. In the first 30 seconds the tension fell to half its maximum value, from 2.1 to 1.05 grams, and thereafter declined more slowly to a level of 0.65 grams. At the end of the five-minute period the stimulation was stopped and the recovery of the muscle was studied by stimulating briefly at five-minute intervals as shown. In the first five minutes the recovery was 90 per cent and in ten minutes it was 96 per cent.

*Discussion.*—The tensions produced by the tensor tympani and stapedius muscles are small in relation to the number of fibers that these muscle contain and the probable strengths of the individual fibers. Casella[3] measured the isometric strengths of single fibers of three kinds of frog muscle and obtained values averaging about 400 milligrams. It is obvious from the results reported here that for both of the tympanic muscles the active fibers are not adding their tensions in any simple way, but to a large extent are working in opposition to one another. This condition has already been suggested by the structural arrangement of the fibers, which is of the pennate type. The effect of a muscle contraction therefore is primarily a fixation of the ligament and only secondarily its linear displacement.

This interpretation of the action of the tympanic muscles is supported by a comparison of the actual effects upon sound transmission with the effects that might be expected simply from the measured tensions. We can calculate these expected effects on the basis of earlier studies by Wever and Bray[4, 5] in which artificial tensions were exerted on the tendons of these muscles. In these studies a thread was tied to the tendon and then led over suitable pulleys to a scale pan. The cochlear potentials were recorded for numerous tones over the range from 100 to 10,000 cycles and the changes in magnitude of the potentials were noted when different weights were placed upon the scale pan. By reference to these data we find, for example, that the average maximum tension of 4.4 grams produced by the tensor tympani muscle decreases the transmission of a 300 cycle tone by 1.6 db, whereas the tension of 1.6 grams produced by the stapedius muscle decreases the transmission of this tone by 5.6 db. The total decrease in transmission afforded by the maximum tension effect of both muscles is thus 7.2 db. Yet we find by a direct measurement of cochlear potentials that the transmission of this tone is reduced by 20 db when (by strong acoustic stimulation of the other ear) a maximum reflex contraction of the muscles is elicited. The difference, which is 12.8 db, is evidently due to a damping action of

the muscles. In the artificial tension studies the thread tied to the tendon is highly flexible in a direction at right angles to its length and thus it will not offer any appreciable resistance to the normal movements of the ossicular chain in response to sounds; the effect in this situation is one of tension only. It is otherwise with the natural mechanism provided by the tympanic muscles. They in their contractions impose upon the ossicular motion a large component of friction along with a moderate increase in tension.

It is plainly advantageous that the ear's protection against excessive sounds depends more upon damping than upon tension, and also that the tensions exerted by the two muscles are partly in opposition, for these conditions give a fixation of the ossicular chain without undue displacement from its normal operating position.

SUMMARY

The strengths of the tympanic muscles were measured by means of a strain gauge when these muscles were stimulated to maximum contraction by electric currents. Large individual differences were found, with means of 3.5 grams for the tensor tympani muscle when connected to the ossicular chain and 4.4 grams when freed of this connection. For the stapedius muscle the means were 1.6 grams for both "restrained" and "free" conditions. Evidence was obtained to show that the primary action of the muscles is to provide a damping of the vibratory motion of the ossicular chain, and their imposing of tension upon this system is only a secondary effect. Also, the tensions imparted by the two muscles are partly in opposition. The result is a protection of the inner ear against overstimulation by sounds with a minimum of displacement of the ossicular mechanism from its normal operating position.

110 ENO HALL.

REFERENCES

1. Lorente de Nó, R.: The Reflex Contractions of the Muscles of the Middle Ear as a Hearing Test in Experimental animals, Trans. Amer. Laryngol. Rhinol. Otol. Soc. 26-42, 1933.

2. Wever, E. G. and Lawrence, M.: Physiological Acoustics, Princeton University Press, 1954.

3. Casella, C.: Tensile Force in Total Striated Muscle, Isolated Fibre and Sarcolemma, Acta Physiol. Scand. 21:380-401, 1950.

4. Wever, E. G. and Bray, C. W.: The Tensor Tympani Muscle and its Relation to Sound Conduction, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:947-961, 1937.

5. Wever, E. G. and Bray, C. W.: The Stapedius Muscle in Relation to Sound Conduction, J. Exper. Psychol. 31:35-43, 1942.

# XLI

# COMBINED EFFECT OF SOUND AND OXYGEN DEPRIVATION UPON COCHLEAR MICROPHONICS IN GUINEA PIGS

JUERGEN TONNDORF, M.D.

REED W. HYDE, CAPTAIN, USAF (MC)

AND

F. A. BROGAN, B.A.

RANDOLPH FIELD, TEXAS

## INTRODUCTION

A survey of the literature shows that extensive studies[1, 10, 13, 16, 17] have been carried out concerning the effects of sound exposure upon cochlear microphonics (CM), both from the standpoint of reversible shifts and of permanent injury. The effects of oxygen deprivation upon the same phenomenon have also been investigated repeatedly[13, 15, 18, 19] and invariably lead to the same conclusion — namely, cochlear microphonics are not affected until oxygen deprivation becomes so severe that breathing and circulation begin to fail. At this point, however, response drops rather rapidly and, unless artificial respiration is administered, levels off at approximately half its original value. From then until after death it declines further but slowly over a period of hours. If the animal is revived in time, the response is quickly restored to the pre-exposure level. This finding seems to put cochlear microphonics in a somewhat unique position with respect to other bioelectrical phenomena, such as nerve action potentials, which are known to decrease in magnitude under moderate degrees of and in gross proportion to oxygen deprivation. This peculiar "nonbiological" behavior had at one time lent support to the so-called "membrane theory" of the origin of cochlear microphonics.[2] This theory assumed that cochlear microphonics arise from vibratory stimulation of the various membranes within the cochlea and, as such, were thought to have no connection with the events leading to excitation of the eighth nerve. Others[13] favored the so-called "hair cell theory." According to this latter theory the origin of cochlear

Dr. Tonndorf is now with the University Hospital, Iowa City, Iowa. Dr Hyde is now with the Victoria Hospital, Montreal, Canada.

microphonics was within the hair cells of the organ of Corti. These theories need be discussed no further since newer experiments[4] have proved that the strongest source of cochlear microphonics (i.e., "first-order CM"), is within the organ of Corti, while microphonics of lesser degree (i.e., "second-order CM"), are produced at various other places within the cochlea (membrane potentials and the like). Apparently, it is these second-order cochlear microphonics which persist after death and have a slow rate of further decay; while the first-order cochlear microphonics quickly recede to a mere fraction of their former value.

Although these findings demonstrate a close relationship between first-order cochlear microphonics and the structures from which excitation of the eighth nerve is commonly thought to arise, the peculiar resistance of cochlear microphonics to oxygen deprivation, as displayed in the aforementioned experiment, is not yet satisfactorily explained. All of these experiments were done in an almost identical manner. The test signals were very weak or they were presented intermittently for just long enough to obtain a response reading. This was done to minimize response shifts which otherwise might be caused by an intense or prolonged test signal.

In line with the concept of stress physiology[12] one might consider this to be a problem of two stresses (sound and oxygen deprivation) having similar effects. It appeared likely, then, that in the absence of one of these stresses (i.e., sound) it would take larger degrees of oxygen deprivation to induce a response shift, but that the combination of both stresses would yield shifts at lesser degrees of oxygen deprivation. Thus a series of experiments was set up in which the combined effect of sound and oxygen deprivation was studied under various combinations of these two stresses.

## CM TESTING PROCEDURE

Guinea pigs were used in these experiments. They were anesthetized by Urethane® (1 gr/kg). Although the effect of Urethane is not as long lasting as that of Dial,® in our experiments, it provided a more relaxed anesthesia, whereas under Dial the animals tended to develop a convulsive type of breathing which markedly interfered with electrical recordings such as that of CM. Sound stimulation both for exposure and for testing was accomplished by a 25-watt horn-speaker (Atlas Sound Corp.) driven by a GR audio generator (type 1301-A) in conjunction with a suitable power amplifier. A plastic tube, two inches in diameter and 36 inches long, was connected to the horn-speaker, and the opening of the tube was placed close to the ear of the animal. Sound levels reported in this paper

are based upon measurements obtained with a sound level meter: The tube connected to the horn-speaker was placed next to the microphone of the sound level meter, instead of to the ear of the animal, and in this manner sound levels were determined. Within the middle-frequency range, undistorted output of this system is in excess of 130 db above standard reference level, which is the highest sound level used in our experiments. From this value the output of the stimulus system could be lowered to any desired level. This was accomplished by placing a Daven attenuator (capable of handling 50 watts) between the speaker and the power amplifier. This equipment had been used in previous similar experiments in this laboratory.[14]

Cochlear microphonics were recorded from a pair of differentiating electrodes (100 M enameled silver wire). The exposed and tapered ends of these electrodes were placed within the two perilymphatic scalae at points in direct opposition across the cochlear duct and close to the round window within the basal turn. This phase of the preparation was greatly facilitated by the use of a specially made burr which was attached to the contra-angle handpiece of a dental drill. An ordinary dental drill was decapitated and provided with a small indentation at its end. A sewing needle, the tip of which was sharpened under the microscope to a fine three-sided point, was mounted in this indentation by means of ordinary lead solder. This served as the drill tip. This method of connecting drill shank and drill tip permitted accurate recentering of the tip whenever it was sharpened. In addition it kept the drill hole from being reamed out, which will invariably occur if very small sized, stiff drills are used, since chucks in dental hand-pieces are not accurately centered.

A reference electrode was placed in the neck muscles. The use of differentiating electrodes in conjunction with push-pull amplification is known to eliminate hum interference and also to eliminate nerve-action potentials which are superimposed upon CM responses in single-electrode pick-up.

A modified Offner preamplifier (type 104-A), a Dumont oscilloscope (type 175-A) and a Brush high-speed-level recorder (model BL-2304) were used for recording purposes. Generally the oscilloscope served as a monitor only, while the voltage level of the response was directly registered by the recorder in db re one microvolt. The automatic registration of the background noise originating within the low-voltage portion of the circuit and affecting the recording of responses to low level signals, was taken during silent intervals between sound signals. This permitted evaluation of the true magnitude of such responses after the experiment was completed. With

this recorder gradient curves of CM responses were recorded for each frequency between the limits of detectability and 130 db of stimulus intensity in 10 db steps within 12 to 15 seconds. Each test signal was applied very briefly (less than one second) so as practically to eliminate response shifts caused by the test signal itself. From registration of such gradient curves in rapid succession, shifts within the linear portion and those of the maximum voltage responses and their respective patterns of recovery could be traced quickly and accurately, so that differences as small as one or two db became meaningful. Since alteration of both of these measures does not always follow an identical pattern, it is important that both of them be registered. It is only lately that this dual registration has been stressed.[10,15]

The method described in the following was used to produce oxygen deprivation. By means of a tracheal cannula, the animal was connected to a wider tube through which air flowed continuously at the rate of eight liters per minute. This tube was provided with three-way cocks so that it could be connected alternately to gas storage tanks which contained various oxygen-nitrogen mixtures. The flow rate within this tube was rapid enough to permit changes to become effective within seconds after the cock was altered. A long connecting line was used between the regulating valve and the point where the tracheal cannula was connected. This was done to keep the animal from perceiving any sound which might be produced by pressure regulation within the system. The outlet, itself, was silenced by a simple muffler which did not create any back-pressure within the system. Because of its length, there was a gradual decrease of pressure within the line so that a manometer inserted close to the regulating valve afforded accurate regulation of the effective pressure at the point where the cannula was connected. This pressure was held to 1 mm of water all during an experiment in order to maintain the animal's respiration in a practically unimpaired state.

In two different series of animals, two oxygen-deficient mixtures were used. These mixtures contained either 10 per cent or 8 per cent oxygen. The actual values of different tank fillings, as checked by a gas analysis (made by Dr. R. T. Clark, Jr., Department of Physiology USAF School of Aviation Medicine), varied between 10.15 and 10.80 per cent in the former and between 8.0 and 8.2 per cent in the latter case.

### EXPERIMENTAL SCHEDULE

As can be seen in Table 1, the experimental schedule was divided into two parts. After the preparation was completed, "normalcy" of CM response was obtained by recording gradient curves for ten

TABLE 1.

EXPERIMENTAL SCHEDULE.

*Part A*: 1. Pretest: Gradient curves for 10 frequencies between 200 and 6000 cps inclusive.

2. Oxygen deprivation (10 or 8 per cent) for 5 minutes during fifth minute, gradient curve for 1000 cps.

*Part B*:

| | Sound exposure period (1000 cps, 130 db, 1 min.) | Recovery period (32 min; gradient curves for 1000 cps repeatedly at log time scale) |
|---|---|---|
| Test 1 | Normal air | Normal air |
| Test 2 | Oxygen deprivation | Oxygen deprivation |
| Test 3 | Oxygen deprivation | Normal air |
| Test 4 | Normal air | Oxygen deprivation |
| Test 5 | Normal air | Normal air |

test frequencies in the range from 200 to 6000 cps (A,1). Further study was limited to one frequency only (1000 cps) since the effect of oxygen deprivation is known to be independent of frequency.[18] Hypoxia was next introduced by letting the animal breathe the oxygen-deficient mixture for five minutes (A,2). During the fifth minute the gradient curve for 1000 cps was again taken in order to ascertain if there was any response shift produced by the oxygen deprivation alone.

The 10 per cent oxygen mixture was used in a series of nine animals. Only three animals completed all tests when the 8 per cent oxygen mixture was used. This was due to the fact that the 8 per cent mixture represents a critical level for guinea pigs and some of them displayed breathing and circulation failure during this test (A,1). When this occurred, these animals were excluded from further testing.

Since the object of this investigation is to detect and differentiate two response shifts, one superimposed upon the other (namely, those induced by exposure to sound and those produced by oxygen deprivation), it was decided to subject each animal to an entire series of tests with and without oxygen deprivation. This was done because in guinea pigs the between-subject variation of response shifts induced

by exposure to sound is known to be so large that reliable data, concerning small additional shifts, could scarcely be gained from between-subject comparisons.

In part B, sound exposure was standardized—i.e., 1000 cps, 130 db above standard reference level for one minute. After each exposure, recovery from the incurred shift was limited to a period of 32 minutes. Gradient curves for 1000 cps were recorded repeatedly during this time at regular intervals which were based on a log time scale, i.e., immediately after exposure, after one-half, one, two, four, etc., minutes. The next exposure was made at the end of this 32-minute period regardless of whether or not recovery was complete. In this manner each animal was subjected to a series of five sound exposures and subsequent recovery periods. In an attempt to differentiate the effect of oxygen deprivation during and after sound exposure, normal air (21 per cent oxygen) and the oxygen deficient mixture were alternately offered as indicated in part B of Table 1. In order to avoid a sequence effect, the actual order of Tests 2, 3, and 4 in part B was varied from animal to animal.

## RESULTS

In Test 2 of part A none of the animals subjected to the entire series of tests showed a shift of the gradient curve during the fifth minute of oxygen deprivation. This was true in both the 10 per cent and the 8 per cent series.

In order to facilitate ready comparison between the test results in part B, these tests were paired for each animal as follows: Tests 1 and 5 (both without oxygen deprivation) served as reference tests in such a way that in the actual order in which they were taken, the second test was plotted with the first one; the fourth test with the fifth one; and the third with the average of first and fifth. Results of these paired comparisons are given in Figures 1 to 4, as response shifts (in db) versus a log scale of time. That is, for the purpose of averaging all animals in each of the two series, the paired tests were arranged in the following manner:

*Paired Tests A.* Oxygen-deficient mixture during and after sound exposure (dashed curve) and the appropriate reference test (solid curve).

*Paired Tests B.* Oxygen-deficient mixture during normal air after sound exposure (dashed curve) and the appropriate reference test (solid curve).

*Paired Tests C.* Normal air during oxygen-deficient mixture after sound exposure (dashed curve) and the appropriate reference test (solid curve).

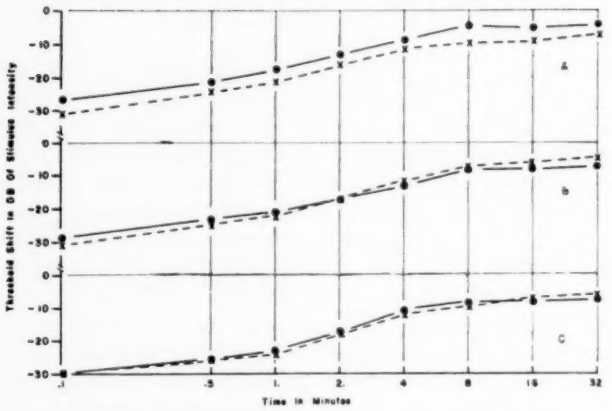



Fig. 1.*—Shift of minimal detectable response within linear portion of gradient curve; 10 per cent oxygen mixture.

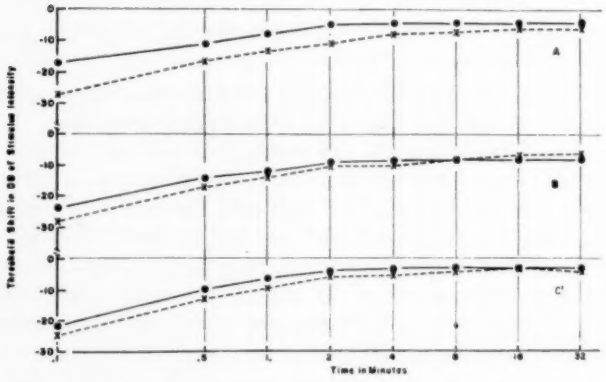



Fig. 2.—Shift of minimal detectable response within linear portion of gradient curve; 8 per cent oxygen mixture.

*Figs. 1 to 4.—Shift of cochlear microphonics vs. time after one-minute exposure to 130 db of a 1000 cps pure tone. Solid curves (in paired tests A through C): Supply of normal air to the animal during and after sound exposure. Broken curves: Paired test A—Oxygen-deficient mixture during and after sound exposure. Paired tests B—Oxygen deficient mixture during, normal air after sound exposure. Paired tests C—Normal air during, oxygen-deficient mixture after sound exposure.

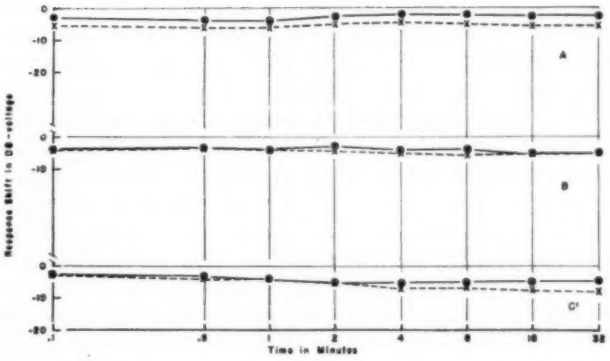



Figure 3.—Shift of maximum voltage response; 10 per cent oxygen mixture.

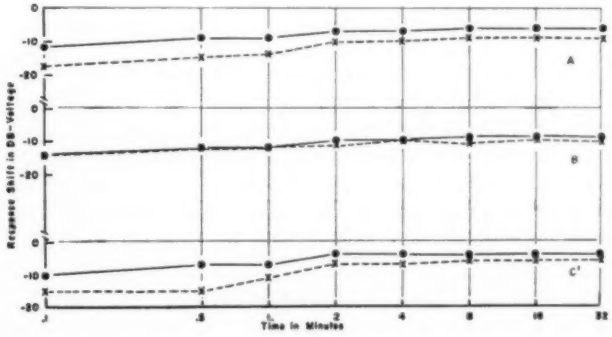



Fig. 4.—Shift of maximum voltage response; 8 per cent oxygen mixture.

These figures confirm the fact repeatedly described by others that recovery of response shifts incurred from exposure to sound approximate a log function of time.

Unless otherwise noted, the differences in the paired comparisons of figures 1 to 4 were statistically quite significant; the t-values being generally well over 3.0. The response shifts within the linear portion of the gradient curve will be described first (Figures 1 and 2). In Experiment A, in which hypoxia was present during both phases, oxygen deprivation was found to induce a larger response shift than sound alone—more so in the 8 per cent than in the 10 per cent series. Since time was given in a log scale, the rate of recovery in the 10 per cent series was delayed, while in the 8 per cent series the recovery is somewhat faster so that it finally approaches the same values found in the 10 per cent series. When normal air was reintroduced at the termination of sound exposure (Experiment B), the initial shift was found similar to that in Experiment A, but recovery took place at a faster rate than it did after sound exposure alone as is signified by the crossing of the two curves in both figures. This increase in the rate of recovery was less pronounced in the 8 per cent series in which, with respect to the 10 per cent series, the crosspoint was shifted toward the right. On the other hand, when oxygen deprivation was present only during the recovery period (Experiment C), there was no effect upon the response. The slight increase in shift during the initial phase of recovery in the 8 per cent series is of questionable statistical significance.

The shift of the maximum output voltages is shown in Figures 3 and 4. As known from previous experience,[15] it was confirmed in the present study that, on the one hand, the initial shift (in db) of the maximum voltage is smaller than that within the linear portion of the gradient curve, and on the other hand, recovery is markedly slower in the former than in the latter case. The additional shift brought about by oxygen deprivation is essentially similar to that within the linear portion of the gradient curve, the only difference being that in Experiment B the two curves do not cross as was the case for those within the linear portion. In Experiment C of the 8 per cent series there is a similar initial shift as seen within the linear portion, although, again, it is of questionable statistical significance.

## PRELIMINARY CONCLUSIONS

From these experiments the following preliminary conclusions can be formulated:

1. The effect of moderate oxygen deprivation upon CM is:
   a. most marked during exposure to sound,
   b. less during recovery from sound exposure,
   c. insignificant with the organ at rest or during minimal exposure, and
   d. related to the degree of oxygen deprivation.
2. The rate of recovery, following combined exposure to sound and oxygen deprivation, was accelerated when normal oxygen supply was restored to the animal.

Although in these experiments an accurate quantitative relationship has not been established thus far between sound intensity and shift of CM, the first point of the above statement may be taken as qualitative evidence of such a relationship.

As to the second part of the statement, it is well known[8] that, as a compensation for the lessened oxygen pressure within the blood, a moderate state of hypoxia is always accompanied by a reactive hyperemia. This hyperemia lasts for a considerable time after the termination of the hypoxia, so that the effective oxygen supply is improved during this period. Therefore, it may safely be assumed that it is this increase in effective oxygen supply to the organ which is responsible for the improved rate of recovery on restoration of normal oxygen supply to the animal.

This increased rate of recovery following restoration to normal oxygen supply was an unexpected finding in view of what is known about the supply route to the organ of Corti, generally thought to be via the stria vascularis and the endolymphatic fluid. Apparently, the effectiveness of this route is not markedly inferior to that of a direct blood supply.

It is not known which of the factors commonly included under the term "hypoxia" might be responsible for these shifts. However, it is very unlikely that they result merely from electrolytic changes brought about in the cochlea, as these should be equal in the presence and absence of sound exposure. In addition, it has been our experience[15] that polarization of the perilymphatic scalae results in shifts of the maximum voltage, but not of the linear portion of the gradient curve. The response shifts described here are linked directly to the oxygen metabolism of the organ of Corti because of (1) the relatively swift reversibility of the shift on restoration of normal blood supply and (2) the dependence of the shift during sound exposure on the degree of oxygen deprivation. Hence, in contrast to our pre-

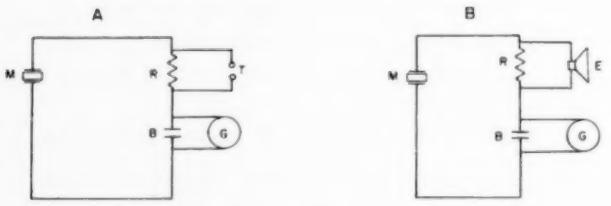



Fig. 5.—Diagram of a carbon microphone circuit (see text).

vious knowledge, the production of CM was shown to depend on the oxygen metabolism of the organ, but only in that the degree of such dependence varied with sound exposure.

## DISCUSSION

In order to discuss the possible significance of these findings, it is necessary to review briefly some general points concerning the principles of microphones. It was stated above that in the production of CM the organ of Corti behaves like a carbon microphone. The basic circuit of such a microphone is given in Figure 5A. In series with a large resistor (R), the microphone (M) is connected to a storage battery (B) which is charged by a generator (G). The changes in resistance produced by vibratory stimulation of such microphones appear as a train of small pulses within the circuit. They can be registered, for instance, by connecting a voltmeter or an oscilloscope at the two terminals (T) across the resistor (R). But as these changes are quite minute with respect to the total resistance, the amount of actual current flow within the circuit is extremely small, that is, expenditure of electrical energy is negligible. This is correct, unless part of this electrical energy is drained from the circuit to perform work—e.g., to drive the membrane of an earphone (E) (Figure 5B). In this latter case electrical energy is expended and this expenditure is in gross proportion to the magnitude of the acoustical force which is driving the microphone.

Actually there is nothing remarkable about the ability of the organ of Corti to produce microphonics. Depending on their mechanical properties, many biological tissues as well as inanimate structures produce microphonics when suitably subjected to vibratory stimulation. The salient question appears to be rather whether or not these microphonic effects actually have a biological function, or in case of the cochlea whether they contribute anything to the

auditory process. This question has been raised repeatedly, but the answer has mostly been negative. However, our findings of energy expenditure in connection with the production of CM may shed new light upon this problem.

To express it simply, the role of the organ of Corti is to convert mechanical vibratory energy, which is transmitted to this organ, into another form which is capable of triggering the nerve response. The possibility that CM may play the role of this required mediator has instrigued many writes,[13] but there never was enough evidence to refute or confirm this hypothesis.

Since there is a period of latency between stimulus and response of the eight nerve and also because of the peculiar frequency relationship between the two, many experimenters felt the necessity for the assumption of a chemical mediator between the sensory cells and the nerve endings in the organ of Corti.[13] To this date no evidence of such an agent has been found. It is felt that the recent discovery of the summation potential[9] has mitigated this difficulty. It was at higher stimulus frequencies where the one-to-one relation between frequency of stimulation and rate of nerve response no longer exists, that this third potential was shown to make its appearance. This mechanism may be likened to that of a condenser with a large time constant so that in cases of higher frequencies it takes more than one period of stimulation to charge it to its full capacity.

Last but not least, it should be pointed out that it is common knowledge among auditory physiologists that the direct application of alternating currents of a given frequency to the cochlea elicits phenomena within the nerve and the higher centers which are identical to those produced by acoustical stimulation of the same frequency. In general, there is more and more evidence that the interaction between neural components is of an electrical nature and that the chemical agents (of which several have been described[13]) represent metabolic products only.[11]

These points, in connection with the evidence presented in this paper, namely, (1) expenditure of energy of CM in gross proportion to the magnitude of sound exposure, and (2) the dependence, within limits, of recovery of CM on the oxygen supply of the organ make it highly probable that CM, as produced within the organ of Corti, are not a mere by-product but constitute the essential link between mechanical stimulation of the cochlea and nervous excitation. The contribution to this process by microphonics produced elsewhere within the cochlea appears unlikely. Because of the high resistance between various locations within the cochlea and the difference in

DC potentials at these locations, such voltages will be attenuated to insignificant values before becoming effective within the organ of Corti proper. This point was given attention in a recent paper by G. von Békésy.[20] It is noted that in this paper a line of reasoning is followed to prove the origination of cochlear microphonics from mechanical stimulation of the organ of Corti, in a manner similar to the one used here, to prove the triggering of nerve action potential by cochlear microphonics.

Because of the complicated electrical circuitry of the cochlea[3] and especially because of the position distances within this circuit of the pick-up electrodes with respect to the organ of Corti, no attempt has been made to present a quantitative evaluation of the data.

## SUMMARY

Gradient curves of cochlear microphonics were recorded from guinea pigs during and after exposure to various combinations of sound (1000 cps, 130 db, 1 min.) and oxygen deprivation (10 per cent or 8 per cent oxygen-nitrogen mixtures). The results indicate that contrary to the concept held previously, CM are affected by oxygen deprivation of moderate degrees, but that this effect depends (1) upon the degree of oxygen deprivation, and (2) on the magnitude of simultaneous exposure to sound. If normal oxygen supply was reintroduced at the termination of sound exposure, the rate of recovery of CM was faster than after sound exposure without return to normal oxygen supply.

In line with the present concept of CM, these findings are taken as evidence that during production of CM and in gross proportion to the magnitude of sound exposure, electrical energy is drained from the cochlea. In accordance with previous evidence, this is thought to indicate that CM, as produced in the organ of Corti, are not mere by-products, but represent the essential link between mechanical stimulation of the cochlea and nervous excitation.

U.S.A.F. School of Aviation Medicine.

## REFERENCES

1. Alexander, I. E. and Githler, F. J.: The Effect of Jet Engine Noise on the Cochlear Response of the Guinea Pig, J. Comp. & Physiol. Psychol. 42:517-525, 1949.

2. Bast, T. H. and Eyster, J. A. E.: In Symposium on Tone Localization in the Cochlea, Annals of Otology, Rhinology and Laryngology 44:792-803, 1935.

3. Békésy, G. von: Coarse Pattern of Electrical Resistance in the Cochlea of the Guinea Pig (Electro-anatomy of the Cochlea), J. Acous. Soc. Am. 23:18-28, 1951.

4. Békésy, G. von: DC Potentials and Energy Balance of the Cochlear Partition, J. Acous. Soc. Am. 23:576-582, 1951.

5. Békésy, G. von: DC Resting Potential Inside the Cochlear Partition, J. Acous. Soc. Am. 24:72-76, 1952.

6. Békésy, G. von: Description of Some Mechanical Properties of the Organ of Corti, J. Acous. Soc. Am. 25:770-786, 1953.

7. Békésy, G. von: Shearing Microphones Produced by Vibration Near the Inner and Outer Hair-cells, J. Acous. Soc. Am. 25:786-790, 1953.

8. Buechner, F.: Allgemeine Pathologie, Muenchen-Berlin: Urban and Schwarzenberg, 1950.

9. Davis, H., Fernandez, C. and McAuliffe, D. R.: The Excitatory Process in the Cochlea, Proc. Nat. Acad. Sc. 35:580-587, 1950.

10. Davis, H., et al.: Acoustic Trauma in the Guinea Pig, J. Acous. Soc. Am. 25:1180-1187, 1953.

11. Marazzi, A. S. and Lorente de Nó, R.: Interaction of Neighboring Fibers in Myelinated Nerve, J. Neurophysiol. 7:83-101, 1944.

12. Selye, H.: Stress, Montreal, Acta, 1950.

13. Stevens, S. S. and Davis, H.: Hearing, New York, J. Wiley & Sons, 1938.

14. Tonndorf, J. and Brogan, F. A.: Procedure for Recording Cochlear Microphonics in Animals, USAF School of Aviation Medicine Project No. 21-27-001, Report No. 5 (Dec.) 1951.

15. Tonndorf, J. and Brogan, F. A.: Two Forms of Change in Cochlear Microphonics, etc., USAF School of Aviation Medicine Project No. 21-27-001, Report No. 6 (July) 1953.

16. Tonndorf, J. and Brogan, F. A.: Cochlear Microphonics Following Noise Exposure, USAF School of Aviation Medicine Project No. 21-27-001, Report No. 7 (July) 1952.

17. Wever, E. G.: Theory of Hearing, New York, J. Wiley & Sons, 1949.

18. Wever, E. J., Lawrence, M., Hemphill, R. W. and Straut, C. B.: Effect of Oxygen Deprivation Upon Cochlear Potentials, Am. J. Physiol. 154:199-208, 1949.

19. Wing, K. G., Harris, J. D., Stover, A. D. and Brouillette, J. H.: Effects of Changes in Arterial Oxygen and Carbon Dioxide Upon Cochlear Microphonics, US Naval Medical Research Laboratory, Report No. 188 (Feb.) 1952.

20. Békésy, Geo. von: Some Electro-mechanical Properties of the Organ of Corti, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 83:448-468 (June) 1954.

XLII

# REPORTS ON THE CHEMICAL COMPOSITION OF THE FLUIDS OF THE LABYRINTH

## IV. FLOW ELASTICITY AND FLOW POTENTIALS IN PERI- AND ENDOLYMPH FROM SHARKS

THURE VILSTRUP, M.D.

C. E. JENSEN, M.S.

AND

JÖRGEN KOEFOED, M.S.

COPENHAGEN, DENMARK

Hyaluronic acid was recently found in the endolymph of acanthias vulgaris.[1, 2] More recently it was discovered that solutions of potassium hyaluronate do not behave like ordinary viscous fluids but show a certain elasticity,[3] which can be demonstrated in a glass capillary with a side hole and an attached syringe. This simple device was invented by Scott-Blair et al.[4] for detection of oestrus in cows through the so-called elastic recoil of the cervical mucus, hence it was named an "oestroscope." Combined with a treatment with hyaluronidase it gives a very sensitive and easy method for the detection of hyaluronic acid.[3]

During a demonstration of the phenomenon of flow elasticity by Jensen and a subsequent discussion with him, the otologist of the present team suggested that the phenomenon might give rise to electrical potentials, and that such potentials might be of profound interest not least in the problems concerning the energy transmission in the labyrinth.

From a physicochemical point of view it is a direct consequence of the model developed by Jensen and Koefoed that such potentials can be expected.

Such potentials could mediate the transformation of mechancial energy into electrical energy (the cochlear microphonic potentials) and they might also provide the adequate stimuli to the sensory cells in the cristae, the maculae and the organ of Corti. The stimulation

From the Universitetes Fysisk-Kemiske Institut, Copenhagen.

could occur from flow potentials either in the endolymph proper or in the gelatinous coverings on the sensory hair cells, e.g. the tectorial membrane and the subcupular protoplasmatic zone around the sensory cells of the crista previously described by Vilstrup.[5, 6]

The actual existence of "flow potential" effects was soon demonstrated on a solution of hyaluronic acid as already reported in a short note.[7]

The results to be reported here are still of a preliminary nature. It appears both from the below mentioned experiments and from the previous flow elasticity measurements that very long relaxation times are involved in the attainment of internal equilibrium in the long chain molecules. The "memory" of past treatment may last for hours and meanwhile chemical changes take place particularly in the endo- and perilymph. Truly reproducible results are therefore difficult if not impossible to obtain and the task of sorting out the various effects of rheological fatigue of coagulation and protein denaturation has been taken up only gropingly.

The potentials were picked up between silver - silver chloride electrodes dipping through the surfaces of the liquid which was placed in a capillary U-tube in which it could be moved backwards and forwards through application of a small pressure difference. Obvious precautions to minimize the influence of noise were taken.

A cathode ray oscillograph was used as an indicator of the potentials and later, when the potential effects had turned out not to be pulses accompanying the movements, but to be positional d.c. potentials of long persistence, a Leeds and Northrup Speedomax potentiometric recorder was applied to increase sensitivity (6 mV full scale range).

Larger potentials than usual were sometimes picked up in the very first displacement with a freshly filled tube and when distilled water was put into the emptied but unrinsed tube. These effects are probably connected to the glass-liquid interface and are therefore better classified as streaming potentials although they are of a more persistent—positional—character than usual streaming potentials.

Flow potentials as a mechanoelectrical effect are related both to piezoelectric potentials and to streaming potentials (frictional electricity). They can for obvious reasons only be picked up at some boundary to a solid phase but their generation is in principle connected to the homogeneous fluid phase in that particular state of strain which reveals itself in the elastic recoil.

## EXPERIMENTS

*Potassium Hyaluronate* solutions, 0.5 per cent, when displaced about 30 mm give potentials of 6-10 mV, as already reported elsewhere. The positive end was at the advancing surface as expected from the theoretical considerations. The elastic recoil of the same solution was 7 mm.

*Endolymph of acanthias vulgaris* was relatively easy to withdraw from the sacculus, but much harder to blow out of the pipette. Similar behavior of endolymph from other animals has repeatedly been reported in the literature. To some extent, at least, it seemed to soften again on standing so as to indicate a workhardening effect. The sharks had been dead for 24 to 40 hours and were sent from Hirtshals in ice.

The pH was 6.6-6.7; the conductivity 0.022 rec. ohm/cm at 22° C.

Recoil of the endolymph was immeasurably small but grew rapidly on incubation with a small amount of hyaluronidase at room temperature as the endolymph became fluid. Later on the recoil disappeared again.

| | | | | |
|---|---|---|---|---|
| After | 15 | min. : | 5 | mm |
| | 30 | " : | 8 | " |
| | 45 | " : | 10 | " |
| | 65 | " : | 5 | " |
| | 75 | " : | 2 | " |
| | 135 | " : | 0 | " |

Hyaluronic acid, hence seems necessary for the ability to recoil and also for gelatination. To cause the latter it cannot, however, be the only active component. To test whether the other component was possibly a protein, trypsin was added to a fresh sample of endolymph and a very remarkable and almost immediate breakdown of the jelly was observed. The relative viscosity of the liquid ten minutes after addition of trypsin was 2.0 (25° C) and during the following hour it decreased to 1.8. The recoil was 12 mm. The trypsin preparation was crystalline Trypure.

*Flow Potentials* of 2 to 6 mV were picked up when thick endolymph with some difficulty was moved through the above-mentioned capillary, but very remarkably with the opposite polarization of hyaluronate solutions, i.e. negative at the advancing surface. Treatment with hyaluronidase made the endolymph easy to handle and

did not, within the first hour, change the ability to give potentials (6 mV were repeatedly recorded), but after several hours the potentials approached zero. Another sample treated with trypsin gave a similar effect, but now the polarity was the same as that of hyaluronate solutions. According to the theoretical considerations this indicates the existence in endolymph of a protein cross-linking which gives to the high molecular lace-work a positive net charge. Liquefaction of the almost gelatinous endolymph can be done either by breaking down the hyaluronate chains or the protein bridges. In the latter case hyaluronate seems to be liberated and its negative charge is allowed to dominate in the high molecular part.

*Perilymph of Acanthias Vulgaris.* Samples from different animals showed a more conspicuous variety than different samples of endolymph.

A few samples were slightly blood-tinged and were immediately discarded.

In some cases we noted a "cloud formation" in the perilymph shortly after the withdrawal and in a very few cases even a coagulation of the whole mass of fluid. These coagula dissolve often spontaneously in the course of about 12 hours. This is strongly accelerated by trypsin. Hyaluronidase, however, has no effect.

These observations indicated the presence in the perilymph of an enzymatic activity. As will be reported elsewhere, a fibrinolytic enzyme has actually been found.

Intermixture of peri- and endolymph is rather easy to avoid during the withdrawal. The sharks had been dead for some time before operation and as the processes of permeation through the membranes in vivo and post mortem are unknown a certain exchange of substances by diffusion cannot be excluded.

The pH values of perilymph were found equal to those of endolymph 6.5 to 6.7. The conductivities were about 10 per cent lower (0.020 rec. ohm/cm). Elastic recoil varied from 4 to 8 mm and potentials from zero to 4 mV with polarity as endolymph. The relative viscosity of a filtered sample, pooled from several animals, was 1.2 at 25° C.

## COMMENT

The mere presence of hyaluronic acid and possibly other mucopolysaccharides as high molecular constituents in the fluids of the inner ear has interesting pathological aspects. The interplay between hyaluronic acid, hyaluronidase and hyaluronidase inhibitors is the

focus of attention in rheumatology, and the possibility that the hydrops-like dilatation of the endolymphatic sacs in patients with Ménière's disease (Mygind[8] and other later observations) could be an osmotic consequence of a depolymerisation of hyaluronic acid deserves attention.

Flow elasticity is a well-known quality among rheologists. Endolymph has hitherto only been described as viscous, but the observation that apparent viscosity is combined with flow elasticity will tend to change the general views on the role of this liquid. As an elastic fluid it will be able to perform small movements without more frictional dissipation of energy than ordinary liquids. The energy will be stored for a considerable length of time as elastic positional energy and regained as kinetic energy when it is allowed to return. A strong damping of possible oscillating movements of endolymph in the cochlear duct has hitherto been presumed, but if mammalian endolymph behaves like that of sharks there is no longer any basis for this assumption.

Flow potentials are, as far as we know, a new departure. Their discovery sets the stage for several stragetic questions concerning the mechanism of the inner ear and will intensify the interest in the electrolytic composition of the labyrinthine fluids.

The potentials which we (on the present basis) can expect to be generated in the labyrinth at moderate degrees of stimulation are probably too small to be in themselves the adequate stimulus of a neuron. We know, however, from the papers by Lowenstein and Sand[9,10] that the action of nerves from the cristae perception is connected to a change in frequency of a "spike potential." For the modulation of a continuous firing[11] it is obviously sufficient with a small potential effect superimposed on the resting potential.*

It is not known and will be very difficult to investigate whether the hair cells in the organ of Corti in a similar way perform continuous random spike-firing which could be then partly synchronized by superposition of an a.c. flow potential on the resting d.c. potential. Co-operation of several firing hair cells should be needed to fire one of the innervating neurons. Tone-localization can now be brought into the picture if two dendrites from one neuron are of different length, so that impulses which are required to co-operate arrive with different time delay at the ganglion. This time delay could vary along the organ of Corti and at the place where this time delay equals the time of swing of a given tone, the neurons will respond to this tone.

*Lowenstein has actually been able to prove that galvanic polarization with currents of only a fraction of a microamp alters the firing frequency, also superimposed on kinetic stimulation.

A more detailed account of these ideas and a discussion of them in the light of the recent literature is under preparation.

SUMMARY

The labyrinthine fluids have been found to have the rheological property of flow elasticity. We have found that this is accompanied by electrical effects, flow potentials.

The numerical values of these potentials turn out to be 2 to 6 mV and 0 to 4 mV for the endo- and perilymph respectively.

The perilymph shows an elastic recoil of 4 to 8 mm, whereas the endolymph shows an elastic recoil only after treatment with hyaluronidase or trypsin.

The conductivity of the endo- and perilymph was measured at 0.022 and 0.020 rec. Ohm/cm at 28° C respectively.

The possible consequences of these observations to the mechanisms of perception in the labyrinth are briefly outlined.

Our sincere thanks are due to the head of this institute Professor J. A. Christiansen and to Professor G. Dohlman, University of Lund, for their kind and stimulating interest in this work.

BLEGDAMSVEJ, 19.

REFERENCES

1. Jensen, C. E.: On Potassium Hyaluronate from the Endolymph of Sharks, Acta Chem. Scand. 8:292, 1954.

2. Vilstrup, Th. and Jensen, C. E.: Determination of Hyaluronic Acid in the Endolymph, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:151, 1954.

3. Jensen, C. E., and Koefoed, Jörgen: Flow Elasticity of Hyaluronate Solutions, J. Colloid Sci. 9:460, 1954.

4. Scott-Blair, G. W., Falley, S. J., Malpress, F. H. and Coppen, F. M. V.: Variations in Certain Properties of Bovine Cervical Mucus During the Oestrus Cycle, Biochem. J. 35:1035, 1941.

5. Vilstrup, Th.: Studies on the Structure and Function of the Semicircular Canals, Monograph, Copenhagen, Munksgaard, 1950.

6. Vilstrup, Th.: Structure and Function of the Membranous Sacs of the Labyrinth in Acanthias Vulgaris, Monograph, Copenhagen, Munksgaard, 1951.

7. Jensen, C. E., Koefoed, Jörgen, and Vilstrup, Th.: Flow Potentials in Hyaluronate Solutions, Nature, in press.

8. Mygind, S. H.: Static Function of the Labyrinth, Acta Oto-Laryng., Supplementum 70, 1948.

9. Lowenstein, O., and Sand, A.: The Individual and Integrated Activity of the Semicircular Canals of the Elasmobranch Labyrinth, J. Physiol. 99:89, 1940.

10. Lowenstein, O., and Sand, A.: The Mechanism of the Semicircular Canal. A Study of the Responses of Single-fibre Preparations to Angular Accelerations and to Rotation at Constant Speed, Proc. Royal Soc. London B. 129:256, 1940.

11. Lowenstein, O.: The Effect of Galvanic Polarization on the Impulse Discharge from Sense Endings in the Isolated Labyrinth of the Thornback Ray. Jour. Physiol. 127:104, 1955.

XLIII

# OBSERVATIONS AND CONSIDERATIONS ON CIGARETTE SMOKING

MERVIN C. MYERSON, M.D.

BEVERLY HILLS, CALIF.

During the past four years there have been a number of statistical studies on the subjects of cancer of the lung. Fourteen studies from six different countries, including the most recent and exhaustive report of Hammond and Horn[1] have established the fact that there is presumptive evidence pointing to a relationship between cancer of the lung and excessive cigarette smoking. In evaluating such studies it should be borne in mind that the number who smoke and contract the disease is a very small proportion of those who smoke and do not develop it. Until the present time no proof has been offered to show a cause and effect relationship between cigarette consumption and this disease. Nor has a carcinogen capable of producing cancer of the lung been isolated from cigarette smoke.[2-5] Despite the statistical significance and apparent relationship between cancer of the lung and smoking, the question of whether smoking is an etiological factor in this disease is yet unanswered.

The author is in agreement with Hueper[6] who feels that if smoking actually plays a role in the production of lung cancer, it seems to be a minor one, if judged from the evidence at hand.

Despite the fact that there are clinical manifestations of the effects of smoking in the larynx and that observations of such effects are quite freqeuntly made by the laryngologist, the incidence of cancer of the larynx has not increased over the years as has cancer of the lung.

Recently the writer removed extensive verrucous leukoplakia from both vocal cords of a 22-year-old male. He had inhaled a concentrated adrenalin solution using a nebulizer several times a day for more than two years. Exhaustive studies of the bronchial tree and lungs failed to reveal any evidence of active disease. Edematous fibroma of the vocal cord, a condition caused by extensive smoking, was described by the writer in 1950.[7] Among almost four hundred

Read before the Postgraduate Assembly, Saint John's Hospital, Santa Monica, California, September 14, 1954.

such lesions which he has seen, none has been known to undergo cancerous changes, yet a similar lesion due to smoking has not yet been encountered in the bronchus.

In the average man, the vocal cords are situated seven inches from the edge of the upper teeth, approximately five to six inches above that portion of the main bronchus most frequently affected by cancer. If an active carcinogen were present in cigarette smoke, one should expect a greater incidence of cancer of the larynx. This is not the case. Since the incidence of cancer of the lung is so much greater than that of cancer of the larynx, it is felt that if cigarette smoke caused changes in the vocal cords, there should be similar evidence of the effects of cigarette smoke in the bronchus. Six of the patients who had been recently operated upon for "smoker's larynx" were bronchoscoped. In none of them was there evidence of significant change in the bronchial mucosa.

Cancer of the lung occurs in non-smokers; it occurs more frequently in urban than in rural areas and varies in frequency in areas of the same type. In any consideration of the subject the relation of smokers to the total population should be kept in mind when discussing the incidence of cancer of the lung. In 1948, Ochsner[8] stated, "in our series we have not been able to show that there has been a higher incidence among smokers than in the population as a whole."

According to Hueper,[6] "the apparent lack of uniformity in human material analyzed by the different authorities is further demonstrated by the appreciable differences in the sex distribution of lung cancer reported at different times by different investigators. The sex ratio fluctuates between two to one and twenty to one. The prevalence of cancer of the lung in males has become more pronounced in recent years. This observation strongly militates against a predominant causal role of cigarette smoking in the production of lung cancer because all previous experience in the field of occupational cancer indicates that given the same type of carcinogenic exposure for both sexes and at the same time an increasing equalization of the intensity of exposure, there occurs a narrowing of the gap in incidence rates of the two sexes and not a widening, which actually exists." The incidence of cancer of the lung in males is much greater than that in females.

Twenty-five years ago cancer of the lung was diagnosed infrequently. The chest specialist, the bronchoscopist and the laboratory have changed this. Today the average patient readily acquiesces to bronchoscopy whereas twenty years ago such was not the case. Cancer of the lung is a disease of the older age group. The mortality rate is

highest among males of the sixty-five to seventy-four group and females seventy-five and over.

Rosenblatt[9] calls attention to the fact that analysis of statistical studies shows very little difference in the smoking habits of the cancer patients and the control groups. Although the percentage of smokers forty to forty-five years of age is very large, the incidence of lung cancer in this group is relatively small.

The effects of excessive cigarette smoking are frequently encountered by the laryngologist. It should be stressed that smoking does not affect the nose or sinuses. One can imagine that these structures could be affected if the smoker deliberately exhaled smoke through his nostrils. Only rarely will the nasal passages show evidence of an allergic reaction to cigarette smoke.

It is not generally understood that after each puff on a cigar, cigarette or pipe, and the emission of smoke, there remains in the oral cavity, pharynx and larynx a residue of smoke. This varies in quantity according to the smoking style or habit of the individual. This residue of smoke which may be supplemented, but is never entirely eliminated during the act of smoking, is the principal factor in the irritation of the local structures. Postnasal drip is encountered in all smokers, regardless of what they smoke. Among cigarette smokers the amount of postnasal drip varies with the type and number of cigarettes smoked and the manner and frequency of smoking. Even a single puff on a cigarette will cause increased mucous secretion in the nasopharynx. One can observe the globules of mucus form in the nasopharynx during the act of smoking when viewing this structure with the nasopharyngoscope.

The uvula, pharynx and larynx show evidence of the effects of excessive smoking. Frequently one sees the submucous space of the uvula filled with fluid. As a result of oft-repeated irritation of the uvula by smoking, this structure presents increased vascularity and firmness. When acutely affected by the irritation of cigar, cigarette or pipe smoke the blood vessels of the uvula become longer and their lumen increase in size. Of interest also is the fact that there is an increase in the number and size of the branches of the main visible vessel immediately after smoking most brands of cigarettes and cigars.

The pharynx appears to be spared in most smokers. Only those who smoke two or more packages a day display increased redness of the postpharyngeal wall.

The most striking changes due to excessive smoking are to be found in the larynx. They range from mild hyperemia to tumor

formation. In some a thickening of both vocal cords is seen while in others whose tissues are more susceptible, there is edema, swelling and fibrosis.

The end result of such changes is an edematous fibroma. The author has previously described this sequence of changes in the vocal cords as a result of excessive smoking and probable increased susceptibility. Of interest is the fact that this condition is seen among cigar smokers as well as cigarette smokers, though not as frequently.

The first manifestation of this condition which has been called "smoker's larynx," is a localized edema of one vocal cord, the only symptom of which is hoarseness. At this stage the edematous swelling will disappear in twelve or twenty-four hours if the individual stops smoking. If he continues to smoke, however, fibrous tissue will be added to the edema. Once fibrosis occurs, the lesion becomes an edematous fibroma and is no longer reversible. Hoarseness is now increased unless the tumor is so situated that it does not interfere with approximation of the vocal cords. It is not unusual to encounter bilateral edematous fibromata without hoarseness because of their location. Among one hundred eighty-two cases operated upon by the author, all but fifteen were male. Six were cigar smokers. All except four of the cigarette smokers consumed two or more packages a day.

In the trachea and bronchi the most frequent effect of excessive smoking is cough. This symptom is more prominent upon arising but may be present during the day and at night, depending upon the quantity and type of cigarettes and cigars smoked. Cough is caused by the accumulation of mucus in the trachea or bronchi as a result of stimulation of the mucous glands. In some cases transient edema occurs which may cause not only cough, but wheezing and dyspnea.[10]

Certain filters and filter tips eliminate a greater part of the irritation of cigarettes. Some are more efficient than others. The adequacy or efficiency of a filter or filter tip may be measured by the density and volume of the smoke which is emitted with each puff. This is because the greater the volume and density of the smoke, the greater the degree of irritation. In addition to filters and filter tips, favorable factors are extra length, lessened intensity of puff, compactness of the tobacco and moisture in the tobacco. Unfavorable influences are increased intensity of puff (inhaling), rapidity of smoking and shortness of the cigarettes.

Physicians are frequently called upon to advise patients concerning smoking because of the conditions for which they are being treated. Experience has shown that a great majority are neither

psychologically nor emotionally equipped to reduce voluntarily the number of cigarettes they smoke. The patient will be served best if smoking is prohibited. Most people reduce the amount of smoking for varying periods of time, but with few exceptions revert to their former smoking habits.

Perhaps that part of this paper which deals with cancer of the lung may best be summarized by referring to the statement of the Minister of Health of England published in the form of a Foreign Letter in The Journal of the American Medical Association, May 1, 1954:

"There has been an increase in deaths from lung cancer in this country which began about 1919 and has continued ever since. The increase is much greater in males than females. Between 1911 and 1919 the number of deaths from cancer of the lungs was about 250 per year. . . . The latest figures available, for 1952, showed an increase in that 11,981 males and 2,237 females died from the disease. The figures also show that the highest mortality rate from lung cancer in males occurred in the sixty-five to seventy-four age group, whereas in females the highest rate occurred in the seventy-five and over age group. Comparable increases have been reported in all countries from which reliable statistics are available.

"Factors such as the increasing age of the population and better diagnosis account for some of the rise but not the whole of it. Tobacco smoking plays some part in this increase. Tobacco smoking cannot be the only factor since the disease occurs in non-smokers. Not one but several factors or a combination of factors must be regarded as responsible. The disease is more prevalent in urban areas than rural areas and different parts of the country suffer more than others. No substance producing cancer of the lungs has yet been specifically identified in tobacco smoke. Certain tars derived from tobacco have produced skin cancers in mice but this is not considered as being conclusive evidence of the presence of a substance producing cancer of the lung."

In conclusion, excessive smoking affects the larynx, pharynx and tracheobronchial tree. The relationship between cancer of the lung and excessive cigarette smoking, though suggested by statistical studies, has not yet been scientifically established.

Efficient filters and filter tips are of value in reducing the irritation caused by smoking.

## REFERENCES

1. Hammond, E. C. and Horn, D.: The Relationship Between Human Smoking Habits and Death Rates, J. A. M. A. 155:1316-1328 (Aug. 7) 1954.

2. Leitch, A.: The Experimental Inquiry Into the Cause of Cancer, Brit. M. J. 2:3262 (July 7) 1923.

3. Cooper, E. A., Lamb, F. W., Sanders, E. and Hirst, E. L.: The Role of Tobacco-Smoking in the Production of Cancer, Jour. of Hygiene 32:(No. 2) (Apr.) 1932.

4. Bogen, E. and Loomis, R. N.: Tobacco Tar: An Experimental Investigation of Its Alleged Carcinogenic Action, Am. J. of Cancer 16:1515-1521, 1932.

5. Wynder, E. L., Graham, E. A. and Croninger, H.: Experimental Production of Cancer with Cigarette Tar, Cancer Research 13:855-864, 1953.

6. Hueper, W. C.: Air Pollution and Cancer of the Lung, Rhode Island Med. J. pp. 34-36 (Jan.) 1953.

7. Myerson, M. C.: Smoker's Larynx: A Clinical Pathological Entity, Am. O. R. L. 59:541-547 (June) 1950.

8. Rigdon, R. H. and Kirchoff, H.: Smoking and Cancer of the Lung—Let's Review the Facts, Tex. Rep. of Biol. and Med. 11(4):715-727, 1953.

9. Rosenblatt, M. B.: Relationship of Smoking to Cancer of the Lung, Presented before Soc. of N. Y. Med. Coll., Mar. 16, 1954.

10. Waldbott, G. L.: Smokers Respiratory Syndrome: A Clinical Entity, J. A. M. A. 152:1390-1400 (Apr. 18) 1953.

XLIV

# PLASTIC ESOPHAGEAL TUBE

WILLIAM W. MONTGOMERY, M.D.

BOSTON, MASS.

The prolonged use of a feeding tube during the period of reconstruction or as terminal care following resection of the cervical esophagus is often a decided problem for the patient and physician. The pleasures of chewing and swallowing become more and more apparent with weeks and months of tube feedings. We have used with success a molded polyethylene tube serving as an artificial cervical esophagus. This paper offers a simple method of constructing such a tube from animal tissue tested polyethylene tubing.

A tube replacing the cervical esophagus must be so constructed as to overcome the following:

1. The upper end of the tube must be molded so that food and liquids will not leak down its sides and also to prevent its sliding down the esophagus during the act of swallowing.

2. The neck of the tube must be so molded that it will not creep up into the pharynx and thus cause gagging and retching.

3. It must be sufficiently long so that food will not be regurgitated up along its sides and out the cervical esophageal opening.

4. The lumen must be of sufficient diameter to allow the passage of well-masticated food.

The basic material for the construction of this cervical esophageal tube is 9.5 mm, inside diameter, 12.5 mm, outside diameter, animal tissue tested polyethylene tubing (Clay and Adams). The length of tubing required to extend from the pharyngeal stoma well down into the thoracic esophagus is about seven to eight inches. The upper end of the tube is constructed as illustrated in Figure 1. (1) indicates the untouched upper end of the tubing. A rubber band is wound around the tube about an inch and a half from its upper end (2). The end of the tubing with the rubber band intact is placed into a beaker of boiling water for about a half minute. When the elasticity of the rubber causes a sufficient constriction of the tubing at this level (3), this end of the tube is placed under cold tap

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

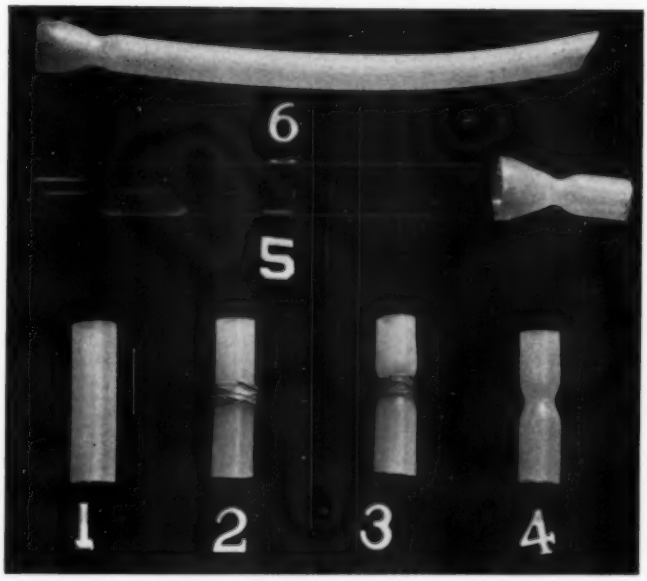



Fig. 1.—(1) Untouched plastic tubing; (2) rubber band in place; (3) after heating and cooling; (4) neck complete; (5) method of dilating upper end; (6) finished esophageal tube.

water. The rubber band is then removed and the molded neck is complete (4). It is obvious that the width of this constricted area is easily variable by the amount of rubber band applied, and the depth of constriction depends on the length of time in boiling water. Test tubes of increasing diameter (larger than the inside diameter of the tubing) are then placed in boiling water. The end of the tubing, above the constricted neck, is placed in the boiling water for a few seconds. The heated base of a test tube with a diameter slightly larger than that of the lumen of the tube is inserted, dilating this end. This is also cooled under tap water, before the test tube is removed. The upper end can then be dilated to any diameter by increasing the size of the heated test tubes. It is wise not to re-insert the once dilated end of the polyethylene tube into boiling water for it will resume its original shape. The diameter of the upper end is thus increased by inserting the heated bases of test tubes and then cooling with water. Both the tubing and test tubes are, of course, handled with towels, during this process. Usually the tubing, when received, has a curve

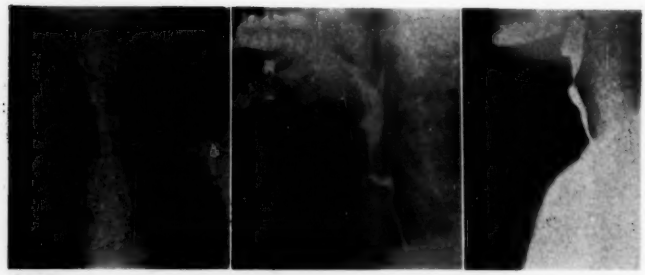


Fig. 2.—A. An anteroposterior view showing the polyethylene tube in place. B. Lateral view taken as a bolus of barium enters the upper end of plastic tube. C. Lateral view demonstrating barium in the pharynx and tube as the patient swallows. The apparent stricture above the tube is merely an evagination of mucosa over the upper rim. This evagination aids in keeping the tube in place when the patient swallows.

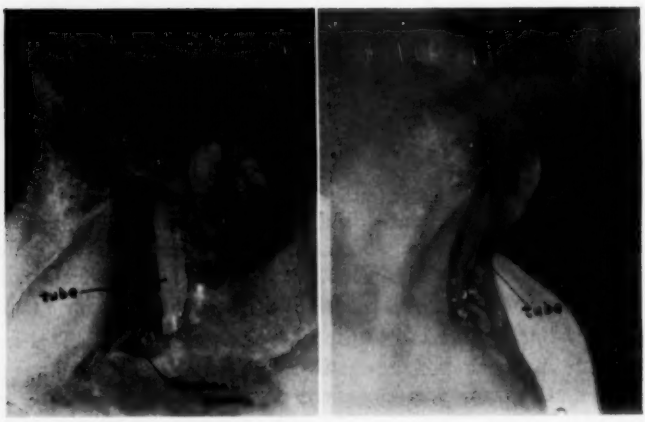



Fig. 3.—Front and right side views of neck with plastic esophageal tube in place.

along its long axis. If the tubing is straight, a slight bend should be made before constructing the upper end. This is done by dipping the whole tube in hot water for a few seconds, bending, and then cooling under tap water. The lower end is tapered to facilitate its insertion.

The pharynx is well anesthetized with 2 per cent pontocaine before the tube is inserted. The molded esophageal tube is grasped in one hand with the index finger in the dilated upper end. It is guided through the upper cervical opening from the pharynx and is then, with the other hand, inserted into the lower esophagus. The molded upper end is then pushed deeply and tightly into the pharyngeal stoma with the index finger. The tube can be removed quite easily and cleaned with a long test-tube brush. This, however, is not necessary if the patient is careful not to swallow large food particles.

We have used a molded polyethylene tube in a patient (Figs. 2 and 3) who has had a total laryngectomy, right radical neck dissection, cervical esophagectomy and extensive radiation therapy. Attempts to reconstruct the cervical esophagus with shoulder flaps have failed because of repeated recurrences of carcinoma. His entire nourishment for more than eleven months was accomplished by the use of the feeding tubes. During this period numerous surgical and radiotherapeutic attempts were made to control the local recurrent disease. After the insertion of this "plastic cervical esophagus" he has received an adequate diet from his own mastication. We have inserted at infrequent intervals a regular feeding tube (for a day or two), with the molded plastic tube in place, as a precautionary measure when the upper pharyngeal stomal area has become irritated. The plastic esophageal tube has also served as a psychological crutch during the patient's progressive illness.

243 CHARLES ST.

XLV

# THE COEXISTENCE OF MALIGNANT TUMORS IN THE TRACHEA AND A DISTAL SECONDARY BRONCHUS

DONALD S. BOLSTAD, M.D.

DETROIT, MICH.

The rarity of separate tumors in the tracheobronchial tree is felt to be of sufficient note to be recorded:

M. L., a white female, aged 55, had been discharged from the hospital in January, 1952, with a diagnosis of right-sided acute bacterial pneumonia, acute pleurisy, and middle lobe syndrome. During her previous hospitalization x-rays had shown a slow clearing of a right middle lobe density. Bronchoscopy had shown bleeding from the right middle lobe bronchus, but no gross lesion was visible. Aspirated secretions examined by the Papanicolaou technique were reported as Class III.

She was readmitted on March 17, 1952, because of increased cough and discomfort in the right chest and beginning atelectasis of the right middle lobe. During the interval since her discharge from the hospital she had gained five pounds.

Bronchoscopy, done the day following admission, revealed a fungating lesion arising from the anterior surface of the trachea, 1 cm proximal to the carina. A separate mass was seen protruding from and causing obstruction to the right middle lobe bronchial orifice. Specimens taken separately from the two lesions were reported as malignant tumors composed of both epidermoid and glandular elements.

On April 2 a right thoracotomy was done, at which time it was planned to perform a right pneumonectomy and resect the lower end of the trachea. The anterior surface of the trachea was carefully inspected. Several glands were removed although no invasion of the trachea, from externally, was seen to have occurred. These glands later were found to have been almost completely replaced by tumor tissue. After transection of the right main bronchus the chest surgeon searched the interior of the trachea for evidence of tumor and

From the Division of Otolaryngology, Henry Ford Hospital, Detroit 2, Mich.

failing to find any gross disease proceeded with the right pneumonectomy. The immediate postoperative course was uneventful and the patient was discharged from the hospital on April 17, fourteen days after operation.

Following her discharge she complained on several occasions of a persistent, dry, hacking cough, usually worse at night, but her weight remained constant and progress chest x-rays showed no change. However, on August 2 a rather marked wheeze was noted and there was some weight loss and increase in cough.

She was readmitted to the hospital and bronchoscopy revealed a large fungating mass arising from the anterior wall of the trachea just above the carina and causing considerable obstruction of the trachea. This was removed almost entirely allowing adequate aeration of the left lung. Biopsy was reported as showing an epidermoid carcinoma with pseudoglandular arrangement and comparison with sections of previously removed biopsy tissue and of the resected lung showed essentially the same pathological picture. At this time it was decided to resort to radiation therapy and she was referred elsewhere for cobalt therapy.

She was given a full course of treatment which apparently controlled the local lesion in the trachea, but she followed a gradual downhill course and, after developing symptoms of cerebral metastases, died in April, 1953.

## COMMENT

Although metastasis of bronchogenic carcinoma to the parenchyma of the opposite lung is not uncommon, only two well-authenticated cases of two concomitant but independent growths in the lower respiratory tract have been found in a search of the medical literature. Bryson and Spencer[1] in their survey of 866 cases of carcinoma of the bronchus, described a case in which an oat-cell and a squamous-cell carcinoma were found in independent bronchi in each lung. Engler[2] reported a case of tumors of the trachea of identical structure, but of separate origin. One tumor was seen to be distinct from the other.

Occurrence of a tumor in the trachea following removal of carcinoma of the larynx 10 and 15 years respectively was described by Orso[3] and Watson.[4] Several instances of in situ carcinoma proximal to the gross lesion in the bronchus were noted in a series reported by Carlisle et al.[5] They also noted that at times a tumor spreading in the extracartilaginous portion of the bronchial wall might push up through the interspaces between the cartilages to form an

endobronchial nodule proximal to the main lesion. In these instances, however, the proximal lesions were separated from the primary tumors by relatively little normal tissue.

Aufses and Neuhoff[6] reported a case in which, five years previously, an apparent "minute carcinoma" had been diagnosed by bronchoscopic biopsy and which, after middle lobectomy and careful pathological study, showed no evidence of tumor. When the patient returned after a five year interval a lesion involving the right main bronchus and extending directly into the trachea was found. Reviewing a case of recurrence of a bronchogenic carcinoma Williams[7] presented evidence indicating that there may be multiple points of origin of bronchogenic carcinoma, especially when it is of the squamous cell variety.

The case here reported demonstrates that lesions may be present simultaneously in portions of the lower repiratory tract, widely enough separated as to be considered two independent tumors. Whether this is a simultaneous development of two distinct tumors of similar structure one cannot say. It is possible that the tracheal lesion may be an implant, inasmuch as it apparently was not present at the time of the first bronchoscopy. Whatever the source, the occurrence of the two lesions is quite unusual.

HENRY FORD HOSPITAL.

REFERENCES

1. Bryson, C. C. and Spencer, H.: Carcinoma of the Bronchus: A Clinical and Pathological Survey of 866 Cases, Quarterly J. of Med. 20:78 (Apr.) 1951.

2. Engler, W. E.: Carcinoma of the Trachea, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 57:429-443 (June) 1948.

3. Orso, L.: Secondary Carcinoma of Thoracic Portion of the Trachea Following Total Extirpation of the Larynx: Case Without Recurrence 10 Years After Endoscopic Operation, Monatschr. f. Ohrenh. 76:584-586 (Dec.) 1942.

4. Watson, W. L.: Cancer of the Trachea 15 Years After Treatment for Carcinoma of the Larynx, J. Thor. Surg. 12:142-150 (Dec.) 1942.

5. Carlysle, J. C., McDonald, J. and Harrington, S. W.: Bronchogenic Squamous Cell Carcinoma, J. Thor. Surg. 22:74-82 (Jan.) 1951.

6. Aufses, A. H. and Neuhoff, H.: Minute Carcinoma of the Major Bronchi: A Follow-up Report, J. Thor. Surg. 23:219-23 (Mar.) 1952.

7. Williams, M. J.: Extensive Carcinoma in Situ in the Bronchial Mucosa Associated with Two Invasive Bronchogenic Carcinomas, Cancer 5:740-747 (July) 1952.

## XLVI

# A REFERENCE POINT SYSTEM FOR INTERPRETING VESTIBULAR TESTS.

A. M. Paisley, M.D.

Jacksonville, Ill.

Since medical school days I have been studying the various tests of labyrinthine function, trying to find some pattern into which all of the various reactions would fit. I felt that one must exist and that it would probably explain the exceptions to our present day labyrinthine laws. I have found that there is, in fact, a definite pattern. Its study has led to the elaboration of a simple system for interpreting vestibular tests. I call it the Reference Point System.

The following phylogenetic approach will make it easier to understand the pattern into which all reactions fall, and therefore the logic behind the Reference Point System.

Eons ago, when the first multicellular organism broke from its coral-like parent to float free on the ocean floor the need for sensing motion came into being. Nature responded to this need by developing, in succeeding generations, a small pit on the dorsal surface of the organism. This pit contained hair cells which were probably activated by sand from the ocean bed and by motion of the surrounding sea water.

In time this pit became closed over and assumed a circular canal shape, the hair cells were surmounted by a gelatinous mass, the cupula, and the canal was filled with a special fluid, the endolymph.

As the organism evolved and developed greater motility there was felt, in the struggle for survival, a need for sensing voluntary as well as involuntary motion. Nature responded to this need by developing a second and later a third canal, until finally our present day vestibular apparatus was evolved, a complete system on either side of the head, both functioning together but each able to take over the duties of the other side.

From earliest time two things were of paramount importance to the organism. One was to obtain food, the other was to keep from becoming food. All sense organs were developed to aid the organism

Read before the Eye, Ear, Nose and Throat Section of the Illinois State Medical Society, May 19, 1954, Chicago.

Fig. 1.—The arrow shows the direction of the endolymph flow in an imaginary resultant canal encircling the head in the frontal plane following either cold irrigation of the right ear or warm irrigation of the left ear with the head erect or following turning to the right with the head 120° forward or turning to the left with the head 60° backward. The nystagmus is in the plane of the resultant canal.

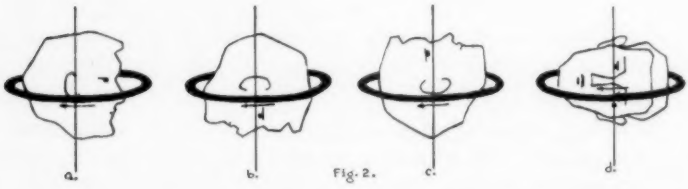



Fig. 2.—The arrows show the direction of the endolymph flow in the resultant following rotation to the right about a vertical axis. With the head upright (30° position) a; head forward (120°) b; head back (60°) c; head 90° on left shoulder d.

Fig. 3.—The right ear is douched with cold water with the head upright (a). The head is then moved into the 120° position (b) and later into the 60° position (c). The arrows show the direction of the endolymph movement. The resultant always remains vertical.

in reaching these two main goals, the food or the haven of refuge. Both goals always lay ahead of the organism and so all senses had as their main function to keep the organism informed as to what lay ahead, and where ahead really was. I could say that the organism was kept properly oriented but here I want to stress the direction ahead.

All end-organs function by sending, under certain conditions, stimuli to the brain. These stimuli are properly integrated and correlated by the brain and certain reflexes are initiated which are always purposive and usually have some protective aspect.

Since the earliest motion-sensing organ was probably a single horizontal canal, I felt that if I were to consider our entire, present day vestibular apparatus as being only one large canal capable of encircling the head in any plane, I might be able to formulate rules that would apply to it.

I could logically do this for it is a well known fact that, after stimulating the labyrinth, we often obtain reactions in a plane which is outside of the plane of any one of our six semicircular canals (Fig. 1 and Fig. 2b, c, d). The nystagmus, for example, may be in a plane which is the resultant of the reactions of two or more canals lying outside of this plane. All reactions are, however, the same as though we actually had stimulated a canal lying in this resultant plane. I therefore called this imaginary canal the "resultant."

I found that this resultant canal in turning tests was always at right angles to the axis of rotation (Fig. 2) and in caloric tests was always vertical (Fig. 3).

Following stimulation by turning, the resultant canal remained fixed to the head as at the time of stimulation, no matter how the head was turned (Fig. 4), but following caloric stimulation it always remained vertical (Fig. 3).

I also found that following labyrinthine stimulation the head and eyes rotated in the direction of the endolymph flow on parallel axes that were always at right angles to the resultant (Fig. 5). Nystagmus and vertigo were always in the plane of the resultant.

I wanted to designate the various labyrinthine phenomena as being in the same direction as or opposite to the direction of the endolymph flow in the resultant canal but I found that in order to do this I must have a point of reference some place on it, a spot toward which or away from which all motion was referred. For example, following rotation to the right with the head erect we would say that the endolymph were flowing clock wise if we were to look down upon the resultant (Fig. 5). But if we were to place a refer-

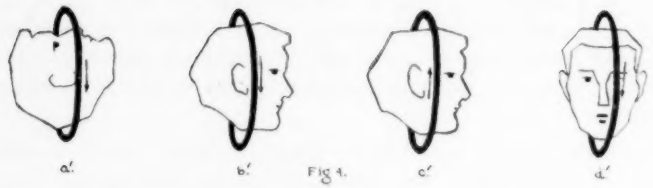



Fig. 4.—Following rotation as in Figure 2, the head is moved to a new position (a in Figure 2 to a' Figure 4 etc.). The resultant maintains the same relationship to the head as at the time of stimulation no matter how the head is moved. Arrows show the direction of endolymph flow which is always the same direction as at the instant following rotation.

Fig. 5.—Following rotation to the right with the head erect the endolymph flows in the direction of the arrow. The head and eyes rotate on parallel axes in this same direction. Looking down upon the resultant we would say that the flow was clockwise. Or we might place a reference point (R.P.) some place on the periphery of the resultant and refer all motion to this point. In the diagram above we would say that the endolymph flowed from left to right past the reference point. A spot C on the eye is closest to the reference point. This spot is drawn slowly to the right in the direction of the endolymph flow and then jumps quickly to the left (nystagmus to the left).

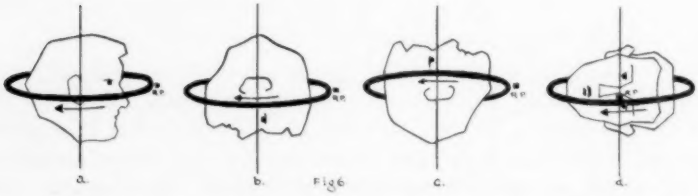



Fig. 6.—Same as Figure 2, but with the reference point properly placed (anterior).

ence point on the anterior half of the resultant we could say that here the endolymph flowed from left to right. When we see that the patient past-points to the right, in the direction of the endolymph flow, and has vertigo and nystagmus to the left, opposite to the direction of the endolymph flow, we know that the reference point is properly placed.

Earlier I stated that I had found a pattern into which all reactions fitted. It has to do with proper placement of the reference point for any given test and is as follows: When the resultant is stimulated in the horizontal plane the reference point is always placed anterior (Fig. 6). Anterior is toward the feet if the person is supine (or rotated with the head back) (Fig. 6 c). If the resultant is in any position other than horizontal we must imagine that first the person and then the resultant are brought into the original phylogenetic position, i.e. horizontal, and then place the reference point anteriorly (Fig. 7). We then imagine the head, the resultant and the reference point brought back to the original testing position (Fig. 7c).

It is important to understand why anterior is toward the feet with the person supine or rotated with the head back 60.° Remember that I stated earlier that all senses, olfactory, visual, tactile, proprioceptive, and vestibular, have since earliest time, had as their main function to keep the organism informed as to what lay ahead and where ahead really was. If an animal running in a given direction slips and falls it does not matter whether it lands prone or somersaults over into the supine position it still knows in which direction it had been running. It still knows the direction ahead, the direction of the reference point. In the supine position this is toward the feet.

Some examples will aid in explaining the use of the reference point:

When we douche the right ear with cold water with the head erect the resultant is of course vertical. Where is the reference point? If we imagine the person on all fours, i.e. ventral surface horizontal, we see that the resultant is also horizontal and we place the reference point anteriorly—in this case over the vertex (Fig. 7). All reactions show that it is properly placed, for the patient's past-pointing is to the right and he tends to fall to the right in the direction of the endolymph flow. His vertigo and nystagmus are to the left, opposite to the endolymph flow. Another example: Rotate a person to the right with the head erect. The resultant is horizontal and the reference point, according to the rule, is to be placed anteriorly, that is, in front of the face.

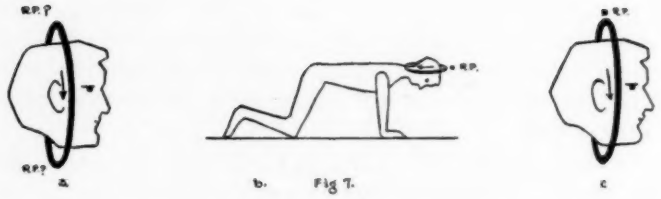


Fig. 7.—Douche the right ear with cold water with the head erect (a). To find the proper position of the reference point we imagine both the person and the resultant brought into the original phylogenetic position, i.e. horizontal, and then place the reference point anteriorly (b). We then imagine the person, the head, the resultant and the reference point brought back to the original testing position (c).

In all routine turning tests the reference point is placed anterior to the horizontally placed resultant at the time of stimulation but following rotation it may change its position if the head be moved. For example: If we rotate a person to the right with the head back 60° the reference point is anterior (Fig. 8a). Following rotation the person past-points to the right and his vertigo is to the left. This is in accordance with an endolymph flow from left to right. If immediately after rotation to the right with the head back we bring the head to the upright 30 degree position we see that the past-pointing changes and is now to the left and that the vertigo also changes and is now to the right (Fig. 8b). According to our rules for placing the reference point we imagine the person on all fours and then bring the resultant into the horizontal position and place the reference point anteriorly (Fig. 7). In the above example this places it over the vertex and we know that it is correctly placed, for only in this half of the resultant (Fig. 8b) is the endolymph flow from right to left in the same direction as the past-pointing and opposite to the vertigo.

So far I have said very little about nystagmus. It is in rotation, with the head back 60 degrees, that we find one of the main exceptions to our present day labyrinthine rules. According to the reference point system following rotation to the right with the head back 60 degrees we elicit a rotary nystagmus to the left, vertigo to the left, and past-pointing to the right.

According to Ewald's laws we should elicit a rotary nystagmus to the right for we have, in this instance, maximally stimulated the right vertical canals. How can we possibly consider Ewald's laws

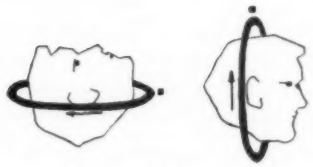



Fig. 8.—To show how the reference point may change its position with change in position of the head. Following rotation to the right with the head back 60° the proper position of the reference point is shown in a. After bringing the head upright the proper position of the reference point is shown in b. Its position in b was determined by applying the rule as illustrated in Figure 7.

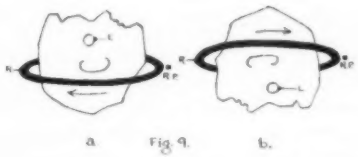



Fig. 9.—Following rotation to the right with the head back 60° (a) the head is moved into the 120° forward position (b). This causes a reversal of the direction of both the past-pointing and the vertigo, although the reflex movements of the eyes do not change in character. However, the direction of the nystagmus is, according to the reference point system, said to change because in each case it is determined by the movement of that spot on the eye closest to the reference point. E-A spot on the limbus at 6 o'clock. L-A spot on the limbus at 12 o'clock. R.P.—Reference Point. R.—Resultant canal. The arrows indicate the direction of the endolymph flow.

to be valid when the patient actually past-points to the right and tells us his vertigo is to the left?

Let us simultaneously turn two patients to the right, the first with the head 120 degrees forward and the second with the head 60 degrees backward (Fig. 2b and c, Fig. 6b and c). After stopping the rotation both patients past-point to the right and both have vertigo to the left. Both should have a rotary nystagmus to the left if we are to be consistent.

According to the reference point system the nystagmus is to the left in both cases but according to Ewald's laws it is to the left only in the patient whose head was 120 degrees forward.

If it is argued that the stimuli in the two cases are diametrically opposite or that it is the changed gravitational pull that accounts for the changes in direction of the nystagmus, logical reasoning would dictate that the direction of the vertigo and past-pointing also must be changed. Only the reference point system adequately explains the phenomena elicited.

If, after turning a person to the right with the head back 60 degrees we immediately rotate the head forward into the 120 degree position we find that there is at no time a change in what is happening to the eyes (Fig. 9). The limbus at 12 o'clock at all times moves slowly to the left and then rapidly back to the right. The person has at all times, according to Ewald's laws, a rotary nystagmus to the right. This certainly cannot be true when vertigo and past-pointing are changed by changing the position of the head. The direction of the nystagmus must also be said to change. According to the reference point system it is said to change. In the first instance the movement of the lower limbus is considered and in the latter instance the movement of the limbus at 12 o'clock is considered. Both are closest to the reference point (Fig. 9).

While I believe that Ewald erred in assuming that a semicircular canal had a unidirectional sensitivity, I believe the greatest mistake was made when rules were elaborated which stated that certain reactions were in the same direction as, or opposite to, the nystagmus. Almost every textbook and article on labyrinthine reactions states that vertigo is to the same side as the nystagmus while past-pointing and head-turning and falling are in the opposite direction.

We must not forget that nystagmus is a reflex, vertigo a sensation depending upon cerebral interpretation of endolymph movement and past-pointing a voluntary motor act and that we will probably err if we formulate rules governing any one reflex, no matter how prominent it is, and then try to fit other reactions into these

Fig 10.

Fig. 10.—Douching the right ear with cold water with a person on all fours causes a horizontal nystagmus to the right and falling also to the right. R.P.—Reference point. R.—Resultant canal.

rules. For example, in the rule above let us consider the statement that falling is in the opposite direction to the nystagmus. Let us douche a patient's right ear with cold water with the head in the 120 degrees forward position and the patient on all fours (Fig. 10). The patient develops a horizontal nystagmus to the right and he falls to the right.

This exception to one of our old, accepted rules is found when the subject is in, what I call, the original phylogenetic position, that is, ventral surface horizontal. Certainly all valid laws should apply to this position!

In using the reference point system we say that the head rotates in the direction of the endolymph flow as it is referred to the reference point, and that falling is to the side to which continued forceful rotation of the head would naturally throw the body.

The literature on labyrinthine physiology is replete with examples showing the need of the reference point system. Consider this interesting passage taken from Jones and Fisher's book "Equilibrium and Vertigo," beginning on page 199: "We always consider vertigo in terms of our relation to what is in front of us, or below us. After the individual has been turned to the right with the head back, the endolymph movement is to the right in relation to objects in front of him. When, however, the head is brought upright, this same endolymph movement is to the left in relation to objects above him. His sensation of vertigo is therefore immediately reversed when the head is brought upright." (Fig. 8)

It is a bit confusing to have some one state that we always refer to objects in front of us or below us and in the next breath that we must now refer to objects above us. The reference point system adequately explains this phenomenon.

Another attempt to explain the reactions following rotation with the head back is found in an article by Benjamin H. Shuster entitled,

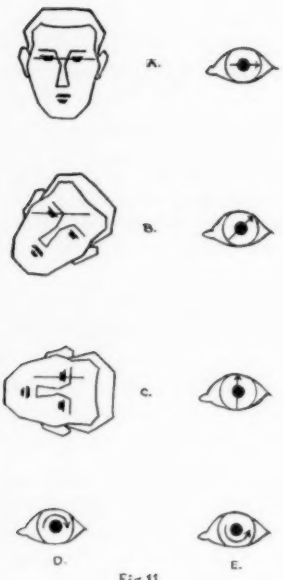



Fig 11.

Fig. 11.—Drawing from Kerrison illustrating Barany's rules for determining in advance the type of nystagmus that should follow rotation to the right with the head in various positions.

"Vertigo," in the October, 1939, Archives of Otolaryngology: "With the head 60 degrees back, when the patient turns to the right the current will flow clockwise in all canals but to the left in the back part of a circle with reference to the patient." (Fig. 9a)

Here Shuster is thinking in terms of a circle, which I call the resultant canal, and is also using a point of reference. He, however, places the reference point posteriorly, which obviously cannot be correct as the patient will tell him that the vertigo is to the left, in the same direction he would ask us to consider the endolymph to be flowing.

It is obvious that Shuster places the reference point posteriorly because he wants the nystagmus to comply with Ewald's laws.

Here we have an example of the fallacy of elaborating rules based upon one phase of a total reaction. It is easy to see how so prominent a reaction as nystagmus tends to dominate the picture and lead us into the pitfalls cited.

Finally I would like to quote from an article by Kerrison entitled "Phenomena of Vestibular Irritation" in the Annals of Otology Rhinology and Laryngology, September, 1909. In it he gives a synopsis of Barany's rules for determining in advance the form of nystagmus following rotation. It shows that Barany really used the reference point system (Fig. 11). "Sitting erect upon a revolving chair a person revolves about a vertical axis. If we now imagine his eye cut through in a horizontal plane i.e., by a plane at right angles to the axis about which he revolves, it is evident that this section will describe a line upon the cornea which will vary according to the position of the head. This line will indicate the form of the nystagmus." After describing various forms of nystagmus he later on says, "With the head bent directly backward so that the face looks upward, a horizontal section describes a curved line below the iris (Fig. 11e). With head in this position, rotation to the right is followed by rotary nystagmus to the left. But in this case the concavity of the nystagmus curve is directed upward." Barany's reference point was the knife sectioning the cornea and he always imagined it brought into the eye in a horizontal position and from a point ahead.

SUMMARY

Our objective is to determine, in advance, the reactions that should follow a given method of stimulating the normal labyrinth.

This objective is attained by imagining the entire labyrinthine apparatus as one large canal around the head (the resultant canal) and then placing an imaginary spot (the reference point) someplace on or near this circular canal to designate *the only point in space toward which, or away from which, all motion is directed.* The movement of only that part of the endolymph, the head, or the eye nearest to the reference point is considered. The position of the resultant canal in any given test is based upon our well-known physical laws. It is horizontal during turning tests and vertical in caloric tests.

The proper placement of a reference point at some point around the periphery of the resultant is determined by applying a simple rule, based upon phylogenesis.

It almost goes without saying that if one knows the normal responses that should follow a given method of labyrinthine stimulation he will recognize any abnormal ones.

209 WEST STATE ST.

## XLVII

# PNEUMATOCELE OF THE TEMPORAL BONE

GEORGE BOTROS, M.D.

CAIRO, EGYPT

In the literature pneumatocele is mentioned rarely in relation with trauma of the temporal bone.

In fractures of the temporal bone escape of air into the tissues may result when the fracture line passes through the air cells by the following routes:

1) Air may pass intracranially into the subdural space, the subarachnoid sulci, the cisternal or brain substance resulting in pneumocephalus as reported by Ruskin.[1]

2) The air may pass between the aditis and the plates of the skull in young adults as reported by Eggston and Wolff.[2]

3) Air may find its way into the extracranial tissues giving rise to surgical emphysema or pneumatocele.[1]

The case reported is concerned with the mechanism of route three.

### REPORT OF A CASE

M. M., aged 48 years, male, complained of swelling on the left side of his head with discomfort on coughing, blowing nose and sneezing, requiring support with the palm of the hand.

Three years previously he was thrown from a car. He fell on the sidewalk on his left side. He was unconscious for six hours. He was diagnosed as a case of concussion and was advised to rest for three weeks. After recovery he resumed work on the third day. He experienced no headache, vomiting, giddiness or tinnitus. He was told by his doctor that little blood came from his left ear.

After one month, he felt with his hand a small painless swelling the size of a small bean behind the left auricle. On pressing it it would disappear to reappear after approximately a quarter of an hour. The primary swelling gradually increased in size.

---

The author is Assistant Professor of Otorhinolaryngology, Faculty of Medicine, Ibrahim Pasha University, Cairo, Egypt.

After one year another swelling began to appear above and in front of the ear. The second swelling was much larger in size at the onset than the first.

Physical examination revealed two swellings on the left side of the head; the first behind the auricle overlying the mastoid part of the occipital bone. It was oval about 6½ x 4 cm. The second in the temporal fossa above and anterior to the auricle was circular like, and approximately 5 cm in diameter.

Both swellings were separate.

Each swelling was soft, fluctuant, non-pulsating with a definite margin. The margin could be rolled under the finger against the bone in the retro-auricular swelling. The margin of the supra-auricular swelling could not be definitely ascertained because of the interposition of the temporal muscle between it and the bone.

There was no crepitus.

Pressure on one swelling caused diminution of its size and increase in the size of the other. Steady pressure on both at the same time for ten minutes resulted in much decrease in the size of both sacs. Positive valsalva made the two swellings reappear again. This emptying and filing of the sacs was not accompanied by headache, giddiness, or tinnitus.

The left auricle and the external meatus were intact.

The upper posterior quadrant of the membrana tympani was thickened and retracted. The tympanic ring at the site was irregular and the irregularity was attributed to previous fracture.

The right auricle, meatus and the membrana tympani were intact. Vestibular tests, caloric: Normal. Both ears.

At this stage the diagnoses entertained were:

1. Meningocele[3] (as it can occur in head injuries of the temporal bone).

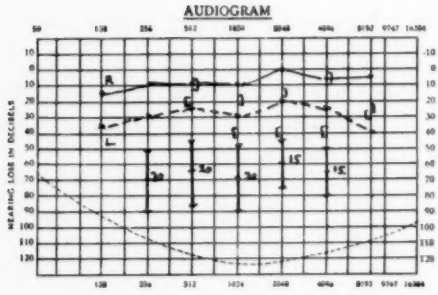
2. Lipoma.

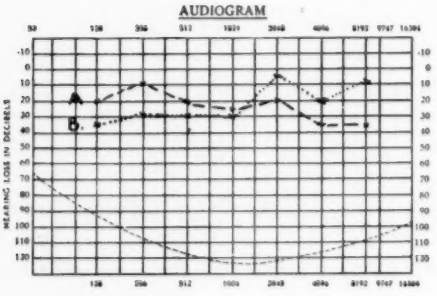
3. Lymphangioma (the margin was ill- defined and infiltrated the scalp).

4. Traumatic implantation dermoid.

5. Epidemoid cyst.[2]

6. Subaponeurotic hematoma (pulsation occurs if there is connection with vessels or sinuses).




Bone conduction in left ear is not of value because of the interposition of the air sac overlying the mastoid.

Lucher's DL is high normal. No cochlear damage.

Audiogram A. Air sacs evacuated by pressure.

Audiogram B. Air sacs filled by positive valsalva.

The increase of pressure raised the hearing in the upper frequencies. The increase of volume diminished the hearing in the lower frequencies. The air cushion in the middle ear is equivalent to a closed chamber of approximately 2 cc in volume.[4] The volume of air varies in different mastoids. In this case the volume of air contained in both sacs having a direct connection with the mastoid cells, middle ear, eustachian tube should have a bearing.

Prior to operation a needle was passed into the posterior sac. Upon aspiration of 45 cc of air both sacs collapsed.

The sac walls were not rigid, and allowed variations in volume and pressure, causing the variations in the audiograms.

*Radiological Examinations:* Views showed highly pneumatized and petrons bones.

The squamosal suprameatal cells extending into the tegmen were well developed.

Right side was normal.

The left mastoid showed destruction of septa, diastasis of the sutures around the mastoid.

The base of skull did not show lines of fractures.

OPERATION

On May 24, 1954, general intratracheal anesthesia of gas oxygen and ether, retroauricular incision was carried through the aponeurosis down to the wall of the post sac. The sac was easily dissected from the scalp and periosteum to one cm of the postmeatal wall. At this point the periosteum was incised and elevated with the sac. A tube of pale bluish epithelium three cm in diameter was seen coming out through a break in the bone in Macewen's triangle to join the sac. This was clamped and the sac removed full of air.

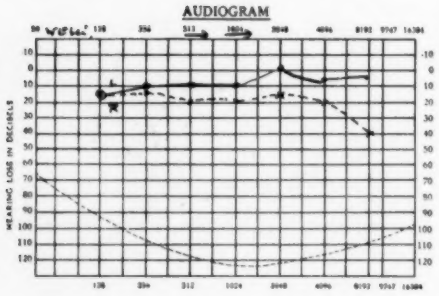
The periosteum was elevated above the meatus over the squama where another tube of epithelium was seen coming through the bone immediately above the suprameatal crest. This tube of epithelium was ruptured and the temporal sac completely collapsed. This sac was not removed.

Each passage in the bone was irregular with sharp edges. Each passed into a very large air cell. The cells were empty with no signs of inflammation. A cortical mastoid operation was done to insure complete obliteration of the mastoid so as to obviate any connection between the air cells, middle ear and extracranial tissues.

The mucosa of the mastoid cells was found shreddy tough, not adherent to the cell walls and did not bleed until the antrum was reached.

The squamous cells were exentrated and brought in one cavity with the mastoid.

Postoperative course was uneventful. August 8, 1954, three months after operation the mastoid had completely healed. The tem-




poral sac which was left was not apparent or felt. Positive valsalva did not fill it.

There is a gain in hearing. There are no dips or peaks. The left ear. Lucher's DL's is high normal. Weber to left with good bone conduction in the right probably exclude retrolabyrinthine damage in left.

The hearing loss can be accounted for by the condition of the tympanic membrane, and the tympanic ring.

The thickness adding mass is affecting the higher frequencies and to a lesser extent the lower tones.

The retraction and the probable fracture of the tympanic ring are increasing the stiffness thus affecting the lower frequencies.

Concussion deafness may give a similar audiogram. Scott Brown[11] reports that concussion is a temporary paralysis of the nerve function and resolves completely in any period up to six months. It is a universal finding that very few cases of concussion deafness become permanent.

*Pathology Report* (by Dr. R. M. Dimmette) revealed:

*Gross:* The formalin fixed tissues measured 5.3 x 3.3 x 2.8 cm. A suture was attached at one point. Near the suture the wall was thicker than at the opposite pole. The wall ranged from 1-4 mm in thickness. The external surface was rough and ragged. A tough white membrane covered one-third of the specimen and muscle was present. The center was cystic and unilocular. The lining was whitish gray and smooth.

Fig. 1.

*Microscopic:* Sections from the wall showed the lining cells to consist of stellate to flattened cells supported by a thick layer of collagenous material. In an occasional site a suggestion of a columnar type pattern was seen. No secretory cells nor true epithelial pattern were seen as that of the middle ear and mastoid cells. This lining was bordered by several planes of loose fibrous connective tissue in which recent hemorrhage had occurred. One layer like that of perosteum was seen in the section taken near the suture. These fibrous layers were bordered by striated muscle which showed little histological alteration.

Diagnosis: Pneumocyst of the scalp; communication with the left middle ear.

Fig. 2.

## DISCUSSION

The patient had two pericranial subaponeurotic air sacs in direct connection with the air in the cells of the temporal bone with two tubes of epithelium passing through two breaks in the bone.

First: How did the breaks in the bone occur?

Second: The formation of the air sacs.

The patient sustained severe trauma to the left side of his head most probably at the back of the mastoid behind the auricle as this escaped injury.

Samuel[5] reported that fracture of the mastoid process was unusual owing to the density of the bone. Fractures involving the mastoid are generally an extension from a fracture of the posterior or middle cranial fossa.

Davis[6] reported that a smashing blow on the mastoid process has been known to fracture the mastoid from the wedge of the petrous bone with injury to lateral sinus.

Ruskin[7] stated that when the force was applied to the mastoid region the fracture followed the occipito-mastoid suture to the jugular foramen, was again continued on the opposite side of the foramen along the petro-occipital suture towards the apex of the petrous

bone. It passed across the sphenoid body to the sphenoid fissure of the opposite side and so across to the anterior fossa.

None of these data can explain the fractures in this case.

The force of the trauma[8] in this case was minimized by the following:

1. The elasticity and resiliency of the large air cells of the highly pneumatized temporal bone.
2. The diastasis that occurred in the sutures.

Keith[9] reported that as late as the 30th year remnants of tectal cartilage may be found between the petro-mastoid and the occipital bones.

Frazer[10] reported that the sutures around the temporal bone begin their fusion at about 30 years of age, complete it in old age; but they seem very variable.

Other factors that may have helped to minimize the trauma:

1. The thickness and mobility of the scalp.
2. Overlapping of the parietal by the squamous bone.
3. The braces of the mastoid and temporal lines.

Thus the patient was saved from severe trauma and probably escaped a fractured base. What happened was that the resilient elastic large air cells of the temporal bone yielded to the force and were compressed. The stress resulted in a bursting fracture with uncapping of cortical cells at two sites. A fracture of the intercellular septa resulted in promoting communication between cells. A fracture of the tympanic ring with rupture of the membrane at the upper posterior quadrant was also present.

The mucosa of the air cells herniated between the edges of the bone at the two sites and prevented the healing of the fractures.

The growth of the sacs was gradual because of the pressure incurred in coughing, blowing the nose, sneezing and straining.

The retroauricular sac was discovered by the patient one month after the accident. The temporal sac was discovered a year later and at a much larger size. This is explained by its relation with the lower transverse fibres of the temporal muscle which kept it in abeyance.

## SUMMARY

A case of trauma to the left temporal bone with development of two subaponeurotic pneumatoceles is reported. One of these was

removed by operation. In this case cortical mastoid stopped connection of the air spaces with the extracranial tissues. Factors of development of the pneumatoceles are discussed.

## REFERENCES

1. Ruskin, S. L.: Functional Anatomy of the Skull, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:1 (Mar.) 1944.

2. Eggston, A. A. and Wolff, D.: Histopathology of the Ear, Nose and Throat, Williams and Wilkins Co., Baltimore, p. 366, 1947.

3. Koch, H.: Acta Otolaryngologica 38:59 (Feb.) 1950.

4. Stevens, S. S. and Davis, H.: Hearing, John Wiley & Sons Inc., p. 262, 1938.

5. Samuel, E.: Clinical Radiology of the Ear, Nose and Throat, H. K. Lewis & Co. Ltd., p. 213, 1952.

6. Davis, E. D.: Discussion on Injury of the Ear Arising from Fracture of the Skull, Proceedings of the Royal Society of Medicine 24:8:1111 (June) 1931.

7. Ruskin, S. L.: The Functional Anatomy of the Skull, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:1:81 (Mar.) 1944.

8. Cordray, D. P.: Basilar Skull Fractures, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 54:3:591 (Sept.) 1950.

9. Keith, A.: Human Embryology and Morphology, Edward Arnold & Co., p. 169, 1933.

10. Frazer, J. E.: The Anatomy of the Human Skeleton, J. A. Churchill, p. 276, 1933.

11. Scott Brown, W. G.: Diseases of the Ear, Nose and Throat, Vol. 2, Butler Worth Co., London, p. 308, 1952.

XLVIII

# VOCAL TECHNIQUE FOR TREATMENT OF CERTAIN SPEECH DIFFICULTIES

Richard O. Bullis, M.D.

and

Alexander Kisselburgh

Los Angeles, Calif.

A major part of speech imperfections—i.e., stuttering, hoarseness, speakers' sore throat, and vocal nodules—is caused by a lack of proper timing of the diaphragm, larynx, tongue, jaw, and lip muscles. Deprived of such subconscious co-ordination, the imperfect speaker submits to the endurance of speech impediments that are well within the reach of non-surgical correction once they are accurately diagnosed.

As a rule, children acquire bad habits of speech by subconsciously imitating the dialect of their parents. Once this dialect has been firmly entrenched in the child's vocal pattern, it is difficult to change or to amend it without concentrated study of the sound vibrations and resonators that should properly be used.

Boland[1] has pointed out that hoarseness may be psychogenic, physiogenic, or both. A causative lesion may be found in the thorax, larynx, pharynx, central nervous system, or other part of the body. Sometimes, he states, no lesion or trauma may be found, and the hoarseness seems closely related to the emotional problems of the patient.

Douglas[2] has grouped the known causes of hoarseness under six main headings: 1) intrinsic lesions, including polyps, contact ulcers, or carcinomas; 2) extrinsic lesions, which produce their symptoms through involvement of the recurrent laryngeal nerve; 3) central lesions, as found in poliomyelitis and myasthenia gravis; 4) peripheral lesions, involving the IX, X and XI cranial nerves; 5) infectious lesions, or those due to tuberculosis, lues, etc.; and 6) psychic factors.

Before embarking on a non-surgical course of therapy, the conscientious physician will, of course, recognize the many and varied causes of speech pathology that may be present. The need for train-

ing in the vocal techniques is greatest when it is obvious that misuse of the voice is at the root of the disorder.

The relationship of voice-production habits to speech impediments and difficulties is evident from the writings of others. Holinger and Johnston[3] have reported a series of 1197 patients with benign tumors of the larynx. Five hundred and twenty-one of them had localized polyps of the vocal cords, and more than 500 of these polyps were attached to the phonating edges of the cords; 193 of the 1197 patients had nodules; and 32 were diagnosed as having granulomas associated with contact ulcers.

The conclusion of these writers is that the influence of vocal abuse as an etiological factor in the development of many of the lesions—particularly the polyps and nodules—was apparent. In 21 cases, previous removal of a polyp had been done previously. It is felt by them that persistence of faulty speech habits after removal of the polyp may be responsible for its recurrence.

Ash and Swartz[4] studied 138 patients made hoarse by "polyps, fibromas, singer's and preacher's nodules, angiomas, amyloid tumors," and other causes and concluded that trauma was an etiological factor. "In practically every one of our cases," they state, "there is a specific history of misuse or excess use of the voice."

Peacher[5] has written that "for the majority of cases [of contact ulcer] surgery, for the most part, is of no avail, since the ulcer frequently recurs after removal. The preferred method of treatment, consequently, should be vocal re-education." The uselessness of imposing enforced silence on the hoarse individual is also stressed by Peacher. She states that "continued misuse of the voice was the perpetuating factor in those patients who still had the symptoms and ulcers after surgery, silence or both. If the patient uses his voice correctly, the amount of talking need not make a difference."

Holinger and Johnston[3] also noted that "voice correction therapy is essential for individuals with nodules and polyps, as well as for those with granulomas associated with contact ulcers."

Through all of the foregoing there runs one thought: that vocal education is often vital in the rehabilitation of speech defects associated with intrinsic lesions and fundamental confusion of voice physiology.

Very few individuals have the gift of perfectly-placed speaking or singing voices. When they do, it is because all of the fundamentals —breathing, throat, lower jaw, tongue, and lip muscles—are co-ordi-

nating with the four resonators which are used to produce the tone. On the other hand, if the tone is directed too much against one resonating chamber (e.g., the nose or the back of the head) it robs the tone of the resonance of other chambers, and part of the tone is thrown back onto the vocal chords.

A prominent university official of Los Angeles suffered throat complications and later developed nodes. His inability to use the proper resonators for a forward direction of tone caused a constant back-lash of vibration in and against the vocal box. A continuous application of vocal exercises, together with proper diaphragm action, soon eliminated all difficulty. This required six months, but to this day, he has not had any recurrent nodes or hoarseness, nor has he resorted to his old custom of speech which had been with him since childhood.

It has been shown that faulty speech can be greatly improved through a knowledge of vocal technique—by both doctor and patient. These muscles must be trained until daily, repeated routine makes correct co-ordination an unalterable habit.

Singing, a prolonged vibration, is intangible. The vocal or spoken word, which is short and tangible, is physical. Vocalization is a matter of physical development. The face muscles, first of all, must be developed, for if the cheeks are relaxed, the tone is pocketed or submerged, while, if the muscles are brought forward by contraction of the lips, the cheeks become taut. The result is that the tone is shot into the nasal resonators, with much the same result as a rifle stabilizing a bullet. Developed muscles are of harder consistency and give more resonance, than undeveloped muscles which are flabby and produce a muted or "mushy" tone.

The therapist must develop the following vocal (or physical) muscles: 1.) Breathing—i.e., diaphragm muscles, abdominal muscles and chest muscles; 2.) throat—i.e., upper (above the epiglottis) and lower (below the epiglottis); 3.) lower jaw; 4.) tongue—i.e., both front and back; 5.) lips.

The latter classification is, obviously, of fundamental importance. The lips are the most forward point of resonance. They must be mobile and constantly moving. If they hang in immobility and muffle the tone, the result—either spoken or sung—is a dead tone. The lips are the megaphone to the voice—a means by which the vowels are colored, not made. Since the corners of the mouth are the pillars of resonance and the trumpet for the tone, their movement should be up and down, rather than sideways. Many individuals

lose a large part of their forward resonance by neglecting the megaphoning capabilities of the lips.

The intangible fundamentals, involved in the prolonged vibrations of singing, are these: 1.) Undertone (body tone); 2.) cut tone (basic mouth tone); 3.) over tone (head tone), which embodies back of the head resonation, nasal resonation, and nostril resonation.

The position of the larynx governs the direction of the vibrations for the desired resonance. A lowered larynx produces a dramatic tone, or one that is rich, full, and powerful. A raised larynx produces a lyric tone, or one that is light and flute-like. It is the latter together with faulty position of the tongue, which causes a great proportion of laryngeal complications.

As an illustration of this point: A Los Angeles executive had three large polyps removed from his vocal cords. After the operation, the inactivity of the larynx caused a constriction of the muscles greatly reducing the opening and, in its raised position, a very high-pitched squeak was the only sound the patient could produce. This condition was overcome by exercising the larynx muscles and lowering the larynx so that in a wider aperture (as in the yawning position) the cords could vibrate in a normal position. This was accomplished in 40 days, and the patient recovered his normal speech.

Light (reedy) vowels are made with the larynx and the speaking muscles. Dark (tubular) vowels are made with the swallowing muscles:

| *Light Vowels* | *Laryngeal Opening* | *Dark Vowels* |
|---|---|---|
| i — sit | Thin | er — sir |
| ee — see | | oo — soon |
| a — say | Wider | oh — bone |
| eh — set | | uh — sun |
| a — sat | | aw — saw |
| ah | Broad | ah |

The identity of all words is established in the front of the mouth, not in the back. It can be said that the identity of the word lies where the consonants are made. When any particular tone is shifted backwards, the vowel sound, hence the syllable, loses its identity, and the word is lost. When vowels are improperly produced, the words cannot be understood. When the tone leaves the area of the lips and teeth, the enunciation loses its clarity.

Enunciation comprises diction, but diction does not necessarily comprise enunciation, which refers to speech and singing. The dictional value of words refers only to the speaking of the words. Character in tone is expressed by the "cut" in the tone, and not by the dominance of the tone. Good public speakers or actors direct their voices forward, with the result that they do not tire easily.

The authors' patients are told to concentrate on the syllabification in the front of the mouth and to open the larynx and arch the diaphragm at the same time. They are also told to keep the tone booming and spinning.

The nomenclature of consonants is as follows:

| Labial (B-P-M-W-F-V) | Lingual (T-D-N-S) |
|---|---|
| Singable | Unsingable or spoken |
| L-M-N-V-M-TH-Z-S | D-G-K-G-B-P-W-F |

When the consonants of a word are emphasized, more of the meaning of the word is expressed. The lack of knowledge in the use of vowels and consonants—especially in the English language—adds greatly to the lack of timing in the use of muscles which are used for each tone. Vowel vibrations are made with the larynx supported by breath controlled by the diaphragm. The vowel sound becomes a spoken or sung word by adding a consonant or consonants. This sound becomes either a dramatic or a lyric vibration, depending upon the position of the larynx.

From the following histories, it is evident that great stress should be placed on the study of vowels and consonants.

1) A 14-year old boy suffered from stuttering and stammering and developed a complex which resulted in general shyness and embarrassment. The developmental theory of stuttering supposes that the stutterer is constituted physiologically and psychologically the same as the non-stutterer, but that, during his development, he has acquired a speech difficulty. This was worsened by social maladjustment resulting from emotional disturbances. The cause of this boy's speech defect was a constriction of the back tongue muscles, which closed the aperture of the larynx and the upper throat. By teaching him, weekly, how to use his tongue and laryngeal muscles, he recovered normal speech in eight weeks.

2) In the case of a motion picture actress, vocal nodes caused irritation, impairing her ability to read her lines. Through proper vocal exercises, she was able to appear in her role in four weeks.

Many common speech difficulties are due to inability to co-ordinate the muscular activity of the vocal apparatus, and can be overcome with proper diagnosis and patience.

727 West 7th St.

REFERENCES

1. Boland, John L., Jr.: The Hoarse Voice. J. Okla. St. M. A. 46:110, 1953.

2. Douglas, T. E.: Hoarseness. Northwest Medicine 49:383, 1950.

3. Holinger, P. H., and Johnston, K. C.: Benign Tumors of the Larynx. Annals of Otology, Rhinology and Laryngology 60:497, 1951.

4. Ash, J. E., and Swartz, Leo: The Laryngeal Node. Tr. Am. Acad. of Otolaryng. and Ophthal. 323, 1944.

5. Peacher, G.: Contact Ulcer of the Larynx. J. of Speech Disorders 12:179, 1947

# The Scientific Papers of the American Laryngological Association

## XLIX

## EVALUATION OF DISSECTION OF THE NECK IN CARCINOMA OF THE LARYNX

Louis H. Clerf, M.D.

St. Petersburg, Fla.

Recurrence following the surgical treatment of carcinoma of the larynx by total laryngectomy commonly is due to an inadequate operation or to undetected preoperative metastasis to regional lymph nodes or to more distant areas. This discussion deals with the problem of block dissection of regional lymph nodes of the neck for clinical metastasis at the time of total laryngectomy, for postoperative metatasis and as an elective procedure.

While the incidence of metastasis in laryngectomized patients varies in different clinics it is generally believed to occur in from 25 to 30 per cent of all cases. This high recurrence rate exerts a profound influence on mortality.

When one recalls that in a majority of cases of carcinoma of the larynx the lesion begins on a vocal cord where lymphatic drainage is scant it would appear that metastasis in this group should be observed infrequently. Too often, however, definitive surgical treatment for the primary carcinoma is not performed until there is subglottic extension or involvement of either the thyroid or arytenoid cartilage. It is generally accepted that in primary carcinoma of the supraglottic and infraglottic regions of the larynx metastasis occurs comparatively early. But these do not constitute a majority of the cases.

The presence of palpable cervical lymph nodes associated with a carcinoma of the larynx should be considered as *prima facie* evidence of metastasis and treated accordingly. In my experience a combined total laryngectomy and complete block dissection have

yielded the best results. Laryngectomy followed by block dissection, or vice versa, after an interval of about ten days is not good cancer surgery. If nodes are observed bilaterally one either can do a bilateral block dissection at the time of the laryngectomy or perform a unilateral resection on the side of greater involvement at the primary operation and treat the opposite side in ten to fourteen days. While these constitute problems in treatment the indications are definite, since metastasis already has occurred.

Of greater importance are the 25 to 30 per cent of patients, who, treated by total laryngectomy, develop regional metastasis postoperatively. It would be a comparatively simple matter if one could institute appropriate treatment as soon as a node is palpated postoperatively. Too often, however, these patients do nothing until the nodes become very large and fixed, or the physician treats them as a simple adenitis for many weeks and then suddenly realizes that they are neoplastic. In the late stages this group usually cannot be treated satisfactorily by any method.

In a series of 285 total laryngectomies previously reported there occurred 67 cases of metastasis to the regional lymph nodes, a percentage rate of 23.[1] Of this group 34 were considered operable and a block dissection was carried out. Nine remained alive and free from carcinoma for five or more years, a survival rate of 26.4 per cent of the operated cases. Of the 33 not treated surgically a number were irradiated but only one survived more than five years without recurrence. That means of the 67 laryngectomized patients who developed metastasis to cervical nodes ten were saved, a total salvage rate of only 15 per cent. It would seem unnecessary to indicate that there was no demonstrable evidence of metastasis at the time of the laryngectomy in these cases, yet in each there was present carcinoma in the regional lymph nodes. If an elective block dissection could have been performed in every case the salvage rate would unquestionably have been much higher.

We are dependent very largely on digital palpation of the neck in our search for nodes. Impalpable nodes cannot be discovered by roentgen studies. A surgical exploration certainly is not warranted. It is imperative, therefore, that a very careful examination of the neck be made before a laryngeal biopsy is done for one often can detect a minute node on the cricothyroid membrane or in the deep cervical chain several days after a biopsy. I agree with Bocca, Ogura and Bello, Orton and others that additional factors must be considered

in a determination of the best plan of surgical treatment of carcinoma of the larynx particularly when the lesion is of long duration or is not limited to a vocal cord and there is an absence of metastasis.[2-4]

A generally accepted principle in cancer surgery holds that one should remove by dissection certain lymph node bearing areas when treating a primary carcinoma on the assumption that these areas are potential sites of metastasis. In practically every field of surgery this principle is accepted. Laryngologists, however, have not generally practiced this and one still observes reports of laryngectomy being performed followed by irradiation therapy to the neck with the hope and expectation that this will destroy a residual carcinoma if present. My experience with irradiation has been disappointing.

An attempt will be made to analyze a series of cases of carcinoma of the larynx treated surgically from 1929 to January 1954 at the Bronchoscopic Clinic, Jefferson Hospital. For comparative purposes the cases will be considered in groups, the first, including all cases treated prior to 1940, then those from 1940 to 1944, from 1945 to 1949 and for a four-year period from 1950 to January 1954.

It will be observed that prior to 1940 there were few patients treated surgically for metastasis to cervical lymph nodes. Total laryngectomy was performed in 90 patients. There were 21 cases of postoperative metastasis to regional lymph nodes. Eight block dissections were performed and three patients were free from recurrence for more than five years, a salvage rate of 14 per cent. During this period one patient with cervical metastasis was treated by laryngectomy followed by block dissection in one week and lived 12 years without demonstrable carcinoma.

In the period 1940 to 1944 combined block dissection and laryngectomy were performed more frequently and elective block dissection with total laryngectomy was started. The first elective dissection was done during February 1942, in the case of a man with subglottic carcinoma of long duration but in whom no lymph nodes could be palpated. He has remained well to date in spite of finding carcinoma in two small lymph nodes along the internal jugular vein.

The number of total laryngectomies during this five year period period was 121. Among these, metastasis to regional lymph nodes occurred in 43. Twenty-eight unilateral and one bilateral block dissection were performed with 14 free from recurrences five or more years. This includes the bilateral case now alive for more than 11

years. Among the irradiated patients one remained free from carcinoma for 10 years. This therefore gives a five-year salvage rate of 35 per cent following surgery and irradiation.

Combined total laryngectomy and unilateral block dissection for demonstrable metastasis were done in three cases with two patients still free from carcinoma.

Laryngectomy and elective block dissection were done in five cases. In all, enlarged lymph nodes were found at operation but carcinoma was demonstrated in but two instances. All remain free from carcinoma.

In the five year period from 1945 to 1949 total laryngectomy was performed in 140 patients. Metastasis to cervical lymph nodes occurred in 49. Secondary block dissection was performed in 32 patients with 19 free from carcinoma after five years. These included five in which bilateral dissection was performed with two alive after five years. Irradiation gave no prolonged benefit.

Total laryngectomy combined with unilateral block dissection for clinical metastasis to the neck was carried out in 15 patients. Six remained free from carcinoma after five years.

Total laryngectomy with elective block dissection were performed in 12 cases. Carcinoma was found in the lymph nodes in four. All are free from any evidence of recurrence after five years.

From 1950 to January 1, 1954, a four-year period, total laryngectomy cases numbered 82. There have been 21 cases of metastasis to regional lymph nodes with 17 treated by unilateral block dissection. Ten of these remain free from carcinoma.

Total laryngectomy combined with block dissection for clinical metastasis was done in 41 cases. In five of these a block dissection was done on the opposite side for metastasis occurring after the first operation. Thirty-five of this group are alive and free from carcinoma. This includes four of the bilateral cases.

Total laryngectomy with elective block dissection was performed in 34. Although enlarged nodes could be demonstrated in all patients at operation, carcinoma in the lymph nodes was found in seven cases. There was one death from pulmonary metastasis. Curiously, there was no carcinoma found in the resected cervical lymph nodes in this case.

Statistics bearing on recurrences and mortality from carcinoma for the latter period (1950-1954) obviously cannot be accepted as final since too little time has elapsed. It is of interest to observe, however, that the number of combined procedures either for clinical metastasis or as an elective operation have increased. They actually constituted 26 and 21.6 per cent respectively or slightly less than one-half of the total number of laryngectomies performed during this four-year period.

All are agreed that in the patient with carcinoma of the larynx and clinical metastasis to cervical lymph nodes, surgical treatment offers more than irradiation in selected cases. The number that can be salvaged is not great, however, irrespective of our surgical approach.

To return to the problem of secondary metastasis to cervical lymph nodes occurring after total laryngectomy, of 351 cases operated upon before January 1950 regional metastasis occurred in 113 (32 per cent). Block dissection was performed in 69 cases (61 per cent). This included six cases of bilateral dissection. There were 36 living and free from carcinoma for five or more years after surgical treatment. In addition one remained free from carcinoma for 10 years following irradiation. It is in this group that our surgical approach must be altered if we wish to improve the five year non-recurrence rate.

On the basis of these statistical data one may assume that in a patient with carcinoma of the larynx with no demonstrable metastasis treated by total laryngectomy only, there is a 25 to 30 per cent chance that there will be metastasis to the cervical lymph nodes. The likelihood that these can be removed surgically is slightly better than 50 per cent and there is a one to two chance that the patient will be free from carcinoma after five years. In this series of 351 cases, 76 (21.6 per cent) of the 113 patients with metastasis to the neck died irrespective of therapy and 37 (10.5 per cent lived for five or more years without recurrence (36 by surgery, one by irradiation).

During the ten-year period from 1940 to 1950 there were performed 17 laryngectomies with elective block dissection. In six patients carcinoma was found in the cervical lymph nodes, although enlarged nodes were found in all cases. There was no operative mor-

tality. There have been no recurrences to date. During that same period there were 92 patients with metastasis to the cervical lymph nodes after total laryngectomy and 58 of these have died.

While the data for 1950-54 cannot be accepted as final they can serve as straws in the wind. There were 21 instances of secondary metastasis to cervical lymph nodes following 82 cases of total laryngectomy, with 11 deaths. One death occurred among the 34 cases of combined laryngectomy and elective block dissection. This proved to be due to pulmonary metastasis. There was no operative mortality and there were no cases of metastasis to the neck.

On the basis of these data it would appear that total laryngectomy and elective block dissection of the neck should be practiced as the surgical procedure of choice in a greater number of cases than heretofore. These should include all cases of supra-and infraglottic lesions unless they are of recent origin and small. While early cordal lesions usually do not metastasize every patient with fixation of an arytenoid or with suggestive subglottic extension should be considered as a candidate for total laryngectomy and elective block dissection.

5575 8TH AVENUE, NORTH.

REFERENCES

1. Clerf, L. H., Putney, F. J. and O'Keefe, J. J.: Carcinoma of the Larynx, Laryngoscope 58:632 (July) 1948.

2. Ogura, J. and Bello, J. A.: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx, Laryngoscope 62:1 (Jan.) 1952.

3. Bocca, E.: Functional Problems Connected with Bilateral Radical Neck Dissection, J. Laryng. and Otol. 67:567 (Sept.) 1953.

4. Orton, H. B.: Evaluation of Radical Neck Dissection, Trans. Am. Acad. Ophthalmol. and Otolaryng. p. 30 (Jan.-Feb.) 1954.

L

# MIXED TUMORS OF THE POSTERIOR PHARYNGEAL WALL

FRED Z. HAVENS, M.D.

AND

LEWELL C. BUTLER, M.D.

ROCHESTER, MINNESOTA

Mixed tumors have long been recognized and it has been noted that they occur predominately in the orofacial region. In 1948 McDonald and I wrote:[1]

"According to Davis, mixed tumors may be defined as 'neoplasms of the orofacial region comprised of stroma of mucoid connective tissue often undergoing cartilaginous transformation with groups of epithelial cells which may be either glandular or squamous.' Bohme stated that mixed tumors occur ten times more frequently in the parotid than in the submaxillary gland and that the sublingual gland is the site of formation of the mixed tumor in one per cent of the whole number. While mixed tumors are common in the parotid gland, they cannot be considered uncommon in the mouth and throat and in the lips. It is exceedingly difficult to trace the genesis of these tumors. In an endeavor to explain the presence of the mesothelial and epithelial elements in mixed tumors, authors have contended that these tumors originate in various ways. There is, however, no unanimity of opinion as to their origin. Volkmann contended that these tumors are of endothelial origin. Krompecher maintained that they are of epithelial origin and that even the cartilage can be derived from the epithelium by metaplasia; however, this has not been considered satisfactory by most authors. The more commonly accepted view is that the cartilage is derived from branchial cleft elements, a hypothesis which was propounded by Cohnheim. McFarland expressed the belief that these tumors originate in sequestrated embryonal material and that some of them originate in branchial cleft elements. . . .

---

From the Mayo Foundation of the Graduate School of the University of Minnesota, Rochester, Minn.

"Possibly we have been unwilling to classify certain lesions as mixed tumor when others might be willing to do so. We have insisted on the presence of two elements; namely, epithelial elements, usually glandular, and myxomatous connective tissue. . . . Squamous epithelium and cartilage are less common components of a mixed tumor, but we have not considered these as essential to the diagnosis."

In the consideration of any neoplasm, careful discrimination on the part of the pathologist is of extreme importance to the surgeon in helping him to decide upon the proper plan of treatment. The mixed tumor belongs to the adenocarcinoma family and in recent years pathologists have learned to discriminate more closely between certain branches of this family. The most notable example of this is the fact that they now separate from the mixed tumors and from the adenocarcinomas the lesion which formerly was placed in the adenocarcinoma group or possibly sometimes in the mixed tumor group but which is now known as cylindroma or, as some prefer to call it, adenocystic carcinoma. (I am inclined to believe that the latter term is the better.) It is noted for its marked tendency to recur in spite of energetic efforts to control it. The adenocarcinomas which those pathologists who employ the grading system will classify as Grades 2, 3, or 4 likewise are difficult to control. They give rise to metastasis and recur more actively and more rapidly than does the mixed tumor if not completely removed.

Pathologists consistently have declined to classify the mixed tumor as malignant, and refer to it as a benign tumor in spite of the fact that it grows relentlessly and recurs persistently if not completely removed. Instances are seen from time to time in which a very active type of adenocarcinoma has arisen from a long-standing mixed tumor. Two cases which illustrate this point come to mind.

CASE 1.—The first is that of a woman who had a tumor of the left submaxillary salivary gland which was known to have been present for 50 years, during which time it had attained a diameter of approximately 4 cm. She sought medical aid because in the previous five months she had noted a small additional nodule developing alongside the 50-year-old mass. Both masses were excised and microscopic examination revealed that the original lesion was a mixed tumor. The new accessory tumor was a Grade 4 adenocarcinoma.

CASE 2.—The other case was that of a man who came under observation in 1922. He had had incomplete removal of a tumor

of the parotid gland a short time previously. On microscopic study of biopsy material obtained from the operating surgeon the lesion was classified as a mixed tumor. This was more than 30 years ago when surgeons were not as bold about undertaking operations on the parotid gland as they are today, and further operative procedures were considered inadvisable. Instead, treatment was given by interstitial irradiation. The patient passed from observation and we know nothing of what transpired in the intervening years, but 30 years later, in 1952, a pathologist wrote that recently he had performed necropsy on this patient and requested information as to the original diagnosis. In return he kindly sent us a slide prepared from an intracranial metastatic lesion and stated that the same type of material had been found in and around the site of the original tumor. Microscopic examination of the slide he had sent revealed a Grade 4 adenocarcinoma.

Observations such as these serve to re-emphasize the necessity for careful and complete removal of all mixed tumors.

## CLINICAL DATA

Mixed tumors occur more frequently in the parotid region than elsewhere, in the submaxillary salivary gland next most frequently, and probably third place is taken by the hard palate where the lesion originates in the general area of the greater palatine foramen. Otherwise such lesions may be found almost anywhere in the orofacial region down to and including the larynx. Among the cases in which the tumor occurs in the latter regions it has been our privilege to see a group of cases in which the tumor seems to follow a very definite pattern as to origin and clinical course. In this group of cases the tumor has had its origin under the mucous membrane of the posterior pharyngeal wall near its lateral limit, apparently at the level of the middle of the tonsil.

As the tumor of the posterior pharyngeal wall enlarges it causes bulging of the adjacent mucous membrane which rarely extends beyond the midpharynx medially but which often extends laterally under the mucous membrane of the lateral wall of the hypopharynx to dislocate the tonsil and sometimes push it over to or beyond the middle of the mouth. Similarly it may extend upward or downward or both. From the clinical description in 39 of our cases we were able to determine that in 18 the tumor had not extended either up into the nasopharynx or down into the hypopharynx; in 9 it had

extended both up and down, in 9 upward only, and in 3 downward only. In some instances the tumor tended to push outward between the mastoid and the ascending ramus of the mandible, and in 7 cases it was palpable externally. This external protrusion may appear as a mass close up under the auricle which may closely resemble a parotid tumor. We have not seen any instance in which there were two separate tumors, therefore, we believe that if the patient has a pharyngeal tumor and also has what might be thought to be a parotid tumor it is best to proceed on the assumption that the mass in the parotid is an extension from the pharyngeal tumor. We have seen primary parotid tumors extend in between the mastoid and ascending ramus but never have seen this extension to be sufficient to produce a pharyngeal mass.

It is the nature of the mixed tumor to grow very, very slowly; consequently, owing to the very slow enlargement the surrounding structures adapt themselves to it, so that rather frequently the patient may be totally unaware of any trouble in his throat until some day he accidentally discovers the tumor and then reports to his physician stating that this swelling has just appeared. (In several of our patients, particularly in the preantibiotic days, the physician who saw the patient under such circumstances had lanced the swelling in the belief it was a peritonsillar abscess.)

We have encountered only two types of lesions that may be mistaken for mixed tumor: lipoma and neurofibroma. Both are slowly growing lesions and either one may remain without symptoms and may become quite large before being discovered. The methods of surgical management recommended for mixed tumors can be and have been employed in the management of either of these lesions.

In our group of cases there were 27 male and 14 female patients. The age distribution is shown in Table 1. As shown in Table 2 these patients presented surprisingly few symptoms.

In 32 instances the clinician recorded his impression as to the consistency of the tumor, as follows: soft 3 cases, rubbery 2, firm 17 and hard 10. The mucous membrane over the tumors was always normal in appearance except when biopsy or "lancing" had recently been carried out.

In 36 of the cases in our series the distribution of the tumors according to the largest dimenison was as follows: less than 4 cm,

TABLE 1

AGE DISTRIBUTION OF PATIENTS

| AGE, YEARS | CASES |
|---|---|
| 20 or less (youngest 16) | 3 |
| 21 to 40 | 11 |
| 41 to 60 | 22 |
| 61 or more (oldest 69) | 5 |

9 cases; 4 to 9 cm, 21 cases; and 10 cm or more (largest 17 cm), 6 cases.

## TREATMENT

Because the mixed tumor cannot be expected to respond satisfactorily to irradiation, the treatment necessarily must be surgical. There are two methods by which surgical removal can be accomplished. One is by an external approach in which a suitable incision is made in the neck so that the external carotid artery first can be ligated after which the tumor is exposed between the angle of the mandible and the adjacent mastoid. These tumors are well encapsulated and the surgeon then can make a blunt dissection of the tumor with his finger. In four of our cases in which this method has been employed the procedure has proved to be very satisfactory.

The other method of removal is to approach the tumor transorally. An incision is made over the most prominent portion of the tumor after which the underlying tissues are separated carefully until the capsule of the tumor has been identified. The finger is then introduced and the tumor is freed by finger dissection. Usually the surgeon's finger is not long enough to reach around the tumor and to get it dissected free completely but usually 80 to 90 per cent of the tumor can be freed in this way. After this has been accomplished an incision is made in the capsule and the margins of it are held by suitable instruments. Through the opening thus made the greater portion of the contents of the capsule can be scooped out. After this has been done the instruments holding the margin of the capsule are rotated to twist the capsule into a cord and with the bulk of the tumor out of the way it usually is not difficult to remove the remainder of the capsule. The cavity remaining after the removal of the

TABLE 2

SYMPTOMS (41 CASES)

| SYMPTOM | | CASES |
|---|---|---|
| "Swelling," "mass" or "tumor" | | 28 |
| Throat | 22 | |
| Parotid region | 5 | |
| Submaxillary region | 1 | |
| Pain | | 5 |
| Throat | 4 | |
| Ear | 1 | |
| None (tumor discovered on routine examination) | | 8 |

tumor is packed loosely with iodoform gauze which is removed in stages beginning after three or four days.

Transoral approach always should be preceded by ligation of the external carotid artery on the side on which the tumor lies. Such ligation may not be necessary but one is taking an unnecessary risk to carry out the operation without it. We know of an instance in which one of these tumors was discovered at the completion of tonsillectomy. The surgeon identified the capsule and thought it would be quite easy to shell out the tumor. He undertook to do so but encountered hemorrhage uncontrollable with the facilities at hand and the patient was lost.

The transoral approach was used in 37 and the external approach in 4 of the 41 cases in our series. The operative results in all 41 cases are shown in Table 3.

In the patient listed as well 11 years after treatment of recurrence the tumor had been removed by the transoral approach. A very small nodule of recurrence had been noted one year after the primary operation and this had been excised.

In the patient listed as well six years after treatment of recurrence the tumor had been removed by the external approach. One year after the primary operation a nodule was found just under the skin at the site of the incision. It was removed and is believed to have been an implantation tumor.

TABLE 3

RESULTS OF TREATMENT

| RESULTS | | CASES |
|---|---|---|
| Postoperative period less than 5 years | | 11 |
| No recurrence | 10 | |
| Recurrence (treated, now well 1 year) | 1 | |
| Postoperative period more than 5 years | | 30 |
| No recurrence (8 had less than 5-year follow-up)* | 24 | |
| No follow-up | 1 | |
| Recurrence | 5 | |
| Treated (well 1, 6 and 11 years respectively) | 3 | |
| Died of disease (tumor not removed) | 2 | |

*Of the eight patients, one died of other disease and without recurrence after 4½ years. The other seven returned for observation at least once, and since they all lived near enough to return easily it is reasonable to assume that none had further trouble.

Four other patients are known to have had recurrence and these will now be reported on in detail, with comments by the senior author.

CASE 3.—A woman, aged 66 years, had a tumor described as as being 10 cm in its greatest dimension. The tumor was explored transorally and found to be partially broken down in its center. Such softening would likely make it much more difficult to carry out a finger dissection of the tumor. We can only speculate as to what probably went through the surgeon's mind; since this was in 1927, before we had such aids as antibiotics and blood banks which now make us unafraid to operate upon the elderly person, presumably the surgeon decided that removal of such a tumor would be unjustifiably hazardous for a woman of 66. At any rate, he made no attempt to remove it but put in radium tubes and followed this with external roentgen-ray treatment. The patient died eight years later, reportedly of "carcinoma of the throat with extension to the cervical nodes." The slides in this case have been reviewed recently and the diagnosis of mixed tumor made 27 years ago has been confirmed.

Metastasis from a mixed tumor is not expected; hence, if the report of "extension to the cervical nodes" is correct, it seems that this mixed tumor probably underwent malignant change in the eight years after operation. Today, under the same circumstances but with all the aids that are now available for the care of the aged, we undoubtedly would go ahead and remove the tumor.

CASE 4.—In this case the surgeon explored the tumor transorally after having made a clinical diagnosis of mixed tumor. On examination of a fresh frozen section the pathologist classified the lesion as a lymphoma. The surgeon, therefore, did not attempt removal but treated the lesion with radium tubes and later radon seeds. It was not until the pathologist had studied the fixed sections that he revised his diagnosis to mixed tumor. This patient, like the previous one, was operated upon some 28 years ago.

Today that same surgeon, because the tumor could be removed readily, would go ahead and remove it in spite of the frozen section diagnosis of lymphoma. In view of the ultimate diagnosis it is obvious that this is what should have been done. The patient lived 13 years. The report of his death clearly indicates that he died of the effects of his tumor.

CASE 5.—In this case the tumor was removed in 1939 by a transoral approach. The patient returned 14 years later with a recurrent tumor that was almost as large as the original. The recurrent tumor was not encapsulated as the original tumor had been, but was made up of multiple nodules. The fact that the recurrence was in the form of multiple nodules made it extremely difficult to be sure of removing all of them. When the patient was re-examined a little more than a year after the second operation there was no evidence of any recurrence.

It is extremely doubtful in this case whether the patient will remain free of disease. There is a definite possibility that the recurrence might have been on the basis of implantation rather than on the basis of failure to remove all of the capsule.

CASE 6.—In the fourth case it seems from the record that when the surgeon made the incision (transoral approach) he must have carried it deeply enough to open the capsule, thus making it impossible for him subsequently to identify the capsule and to dissect it properly and remove it. This patient returned nearly three years

later with a very small nodule of recurrence and this was treated by interstitial irradiation.

It is doubtful whether this patient's tumor will be controlled. It is extremely important that the capsule be identified in order that the dissection can be made with a blunt instrument or with the finger.

## SUMMARY

Mixed tumor is rarely found in the posterior pharyngeal wall. Our survey covered a period of 37 years, during which there were 1,500,000 clinic registrations.

In the literature there are scattered reports of similar cases but it is difficult to formulate a firm policy regarding the management of any particular type of lesion from a study of isolated case reports. We hope that this survey and its analysis will help in formulating definite principles in connection with the management of these lesions.

Although we believe that treatment should be surgical, it may be immaterial whether the surgeon chooses to remove the tumor by the external or by the transoral approach. There is, however, one thought that might be taken into consideration in deciding which approach to use. Since these tumors usually are large and since often it is necessary to open the capsule and remove part of its contents before the remainder of the capsule can be removed, there is of course the danger that recurrence might be brought about by "seeding" (implantation of fragments of tumors). Our records indicate that "seeding" was believed to have occurred in one of four cases in which the tumor was removed by the external approach, whereas it was believed to have occurred in only one of 35 cases in which the tumor was removed by the transoral approach. An explanation for this difference might be that a clean surgical wound that is closed as a neck wound is closed would provide much better soil for possible "seeding" than would a wound which is packed wide open with iodoform gauze and which is left in place for four to seven days.

MAYO CLINIC.

## REFERENCE

1. McDonald, J. R. and Havens, F. Z.: A Study of Malignant Tumors of Glandular Nature Found in the Nose, Throat and Mouth. S. Clin. North America 1087-1106 (Aug.) 1948.

LI

# VASOMOTOR RHINITIS

THEO. E. WALSH, M.D.

ST. LOUIS, MO.

Although the literature of the past century abounds with articles on vasomotor rhinitis, the sufferers from this complaint remain some of the rhinologist's most distressing problems. It is the purpose of this paper to discuss the etiology and those contributory factors so important in the disease, and to invite discussion regarding the principles of therapy which may be expected to give some measure of relief to our patients.

The condition under discussion is that which is characterized by constant nasal stuffiness and increased postnasal discharge, accompanied by dull headache and a feeling of fatigue and nervousness. The common findings are a swelling of the turbinal mucosa with glairy mucus in the meatuses, a peculiar red or purplish color of the turbinates and excessive lymphoid nodules on the posterior pharyngeal wall and in the nasopharynx. In some instances there may not be found an excessive secretion in the nose and when smears of nasal secretions are obtained they show few cells, and eosinophilia is not usual except during exacerbations of the disease.

## ETIOLOGY

If one accepts the definition of allergy as "the acquired specific altered capacity to react" then vasomotor rhinitis and allergic rhinitis are synonymous. By common usage, however, the term allergic is applied to those cases, exemplified by hay fever, in which there is an acute reaction characterized by vasodilatation, edema, excessive secretion of mucus and the presence of eosinophilic granulocytes in the secretions and tissues. This allergic state is the result of an antigen-antibody reaction in which there is a release of histamine with a resulting capillary dilatation, an increased capillary permeability and consequent tissue edema.

The problem of the acute allergic nose can be solved when the antigen is recognized and can be removed from the patient's environ-

ment or when he can be treated with a series of hyposensitizing injections. The problem of the vasomotor nose is much more difficult because it is rare to be able to find a causative allergen either from a careful history or from skin tests. There is one type of case, however, that is probably truly allergic. It is exemplified by the child with vasomotor rhinitis usually bright, intelligent and spoiled whose mother informs the physician that the child eats "hardly anything" and only "picks at his meals." Yet it is obvious that the child is not undernourished. If one is able to speak to such a child by himself, one can usually find out that although he eats little at meal times he is constantly eating candy bars and drinking Coca Cola between meals. It is not only in children that one sees such dietary indiscretions but also in adults. The resultant vasomotor rhinitis can be corrected if a properly balanced diet is followed. Dohlman[1] emphasized dietary indiscretions as a cause for vasomotor rhinitis and noted that in his patients it was usually a diet predominately carbohydrate which caused the trouble. He, too, found that when the diet was regulated and properly balanced the vasomotor symptoms in the nose disappeared.

There can be little doubt that endocrine dysfunction may cause nasal congestion. Swelling of the nasal tissues is not an uncommon accompaniment of the menses and all of us are familiar with the nasal congestion which occurs in some women during the last weeks of pregnancy. Proetz[2] in 1947 discussed the influence of the thyroid in nasal disease. Most of his cases were of the hypothyroid type having basal metabolic rates of less than —11 per cent. A few cases that he reported had increased basal metabolic rates. Laub[3] in 1945 and Walsh[4] in 1950 pointed out the influence of the endocrines on the nose, not only of the thyroid but of ovarian and testicular hormones. Both quote cases in which the nasal symptoms were relieved by the use of estrogens or androgens.

It is generally realized that emotional states may lead to peripheral vascular change. For example, the blanching that occurs with fear; the blushing with shame or embarrassment. Wolff[5] and his co-workers have pointed out the influence of emotional disturbances on the function of the nose. They have even shown that emotional disturbances may lead to chronic organic changes in the nasal mucosa and that acute emotional disturbances may cause eosinophilia in tissues and in the peripheral blood.

The question arises: Are the psychosomatic and endocrine factors operative only in those individuals who are truly allergic or are these aberrations themselves the cause of the altered nasal state? Perhaps, the question is purely academic. In Proetz' series less than 15 per cent were demonstrably allergic. I have been unable to demonstrate an allergy in most of my patients who have responded to endocrine therapy. I believe, however, that in most instances it is probably the addition of psychic or endocrine insult to the injury, however mild, of allergy that causes trouble.

## MANAGEMENT

The objectives of management are surely, 1) to determine and treat the cause of the vasomotor state and, 2) to provide the patient with some measure of relief during such investigation. In regard to the provision of relief for the patient, I believe the use of vasoconstrictors as nasal drops or sprays cannot be too highly condemned. It is probable that clinical investigation of a patient with vasomotor rhinitis will take time. The effect of a local vasoconstrictor in the nose is very temporary and continued use leads to turgescence. This is especially true of Privine® but I believe all local applications can become a habit and can themselves lead to a congested state indistinguishable from the condition for which they were prescribed. Relief of nasal congestion can be afforded by the use of vasoconstrictors by mouth and I have found Propadrine® 3/8 gr useful for this. Occasionally its effect is enhanced when it is combined with an antihistaminic. The importance of a careful clinical history need hardly be emphasized here. Knowledge of the patient as a whole person is essential in evaluating his case. The greatest criticism of the specialist is that he is apt to concentrate on his particular field and forget that he is primarily a physician. A suggestive allergic history should be investigated by appropriate skin tests and by the regulation of the diet. In those instances in which the thyroid is suspected, I have found that the patients exhibiting a low basal metabolic rate with a high blood cholesterol have responded best to thyroid medication. In regard to the dosage, I think it is important that sufficient thyroid extract be given to be of use. It has been our routine to start the patient with one grain a day for perhaps two weeks, and then increase the dose to two grains a day for two weeks, then three grains a day for the next two weeks and so on until changes in the nasal mucosa can be observed. We have found in some instances

that as high a dose as four or five grains a day is necessary. We have seldom observed changes in the basal metabolic rate even with high doses. After mucosal changes have occurred we diminish the dose until an adequate maintenance dose is found. We have also found that the use of vitamins with the thyroid has been helpful and we have suggested that 50 mgs of thiamine chloride and 0.5 gm of ascorbic acid a day is of benefit.

For those patients whose emotional problems seem to be of paramount importance, I believe that a sympathetic ear and human understanding is as useful in an otolaryngologist as in a psychiatrist. The patient should be allowed sufficient time to avail himself of both. I have found it very difficult to persuade patients to see a psychiatrist and equally difficult to interest the psychiatrists in these patients. Consultation with an internist, allergist and endocrinologist is in the best interest of the patient. That the very small doses of histamine that Hansel[6] advocates will cause amelioration of symptoms in some cases cannot be denied. I confess, however, that I cannot understand why this should be so.

SUMMARY

1. Endocrine, dietary and psychosomatic disturbances play an important part in the etiology of vasomotor rhinitis.

2. Allergic factors are not identifiable in a large percentage of cases.

3. The use of local vasoconstrictors or drops or sprays is inadvisable in the symptomatic treatment.

640 S. KINGSHIGHWAY.

REFERENCES

1. Dohlman, G.: Personal Communication.

2. Proctz, A. W.: The Thyroid and the Nose, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 56:328, 1948.

3. Laub, G. R.: Rhinitis Vasomotoria Due to Imbalance of the Endocrine Glands, Laryngoscope 55:479.

4. Walsh, T. E.: Vasomotor Rhinitis, Laryngoscope 60:360.

5. Holmes, T. H., Goodall, H., Wolf, S. and Wolff, H. G.: The Nose, C. Thomas Co., 1950.

6. Hansel, F. K.: The Treatment of Certain Specific Types of Headache with Histamine, Trans. Am. Laryng. Rhin. & Otol. Soc., 228, 1947.

LII

# THE RADICAL OBLITERATIVE FRONTAL SINUS OPERATION

## A CONSIDERATION OF TECHNICAL FACTORS IN DIFFICULT CASES

ROBERT L. GOODALE, M.D.

BOSTON, MASS.

The purpose of this paper is to call attention to some of the problems which the author has encountered in dealing with severe cases of frontal sinusitis. These cases are those in which complications exist so that surgical procedures such as the Jansen-Lynch-Smith operation are not adequate. They fall into three general groups: First, traumatic; second, chronic osteomyelitic; and third, cases which have had repeated operative failure.

The procedure which I am considering today is not by any means a new one. It actually antedates most of the present day operative techniques. At the Massachusetts Eye and Ear Infirmary, it is called the "Obliterative Frontal Sinus Operation." As a matter of fact obliteration was first attempted by Kuhnt[1] prior to 1895. At this operation, the anterior wall was removed but the floor of the frontal sinus was allowed to remain, the ethmoid cells were undisturbed and an attempt was made to maintain drainage and to administer medication by means of a temporary rubber drainage tube placed in the external incision. Riedel[3] later removed not only the anterior but the inferior wall. Ropke[2] modified the operation further by including exenteration of the anterior ethmoidal cells thus establishing drainage into the nose. The need for external drainage was thus eliminated. While all of these operations attempted to obliterate the sinus so that there would no longer be any air space and no longer any mucous membrane which could later become reinfected, special attention was not paid to the role of the ethmoid cells. In my opinion, to perform this operation properly, not only the anterior wall and the floor of the frontal sinus should be removed completely so

that there is no longer a vestige of cavity in the frontal area, but the ethmoid labyrinth should also be completely exenterated, as it is by way of the ethmoid that reinfection will take place. All the partial septa of the frontal sinus and all of the mucous membrane should be meticulously removed. Leaving small areas of mucous membrane and small unobliterated spaces in the frontal area lead inevitably to formation of mucoceles. As you can see, this operation allows the soft tissues of the forehead including the periosteum which originally covered the anterior wall of the frontal bone to come in contact with the denuded posterior wall of the frontal sinus. This union obliterates the air space of the frontal sinus.

In a previous paper, the author analyzed the cause for failure in frontal sinus surgery as found at re-operation of cases done at the Massachusettes Eye and Ear Infirmary. Most of these cases, to be sure, were of the Lynch type. The failures were due to the fact that 1) the entire floor of the frontal sinus was not removed, 2) partial septa were allowed to remain, 3) the operations had failed to remove all the ethmoid cells and, 4) obstructing adhesions sprang up in the nasofrontal duct area.

In a case requiring obliteration of the frontal sinus, it is no longer desirable or possible to maintain a nasofrontal duct but it is just as important to remove the entire floor of the frontal sinus and to complete the ethmoidectomy. In addition, it is absolutely necessary to curette away every vestige of mucous membrane from the posterior wall and to base-level every partial septum with painstaking thoroughness. I mention these points again because in several of the cases I am going to cite, partway measures were attempted at previous operation with the result that the obliterated sinus developed mucoceles or pyoceles and thus became severely reinfected.

When do we remove the posterior wall? Dr. Mosher advocated the routine removal of the posterior wall. In cases where the bone is grossly diseased I would agree with this, but in practice I believe that where the posterior wall is solid and healthy and there is no suspicion of intracranial disease, all that is required is the most careful removal of the mucous membrane.

In essence, this operation calls for no greater technical skill than that which we are accustomed to use in other radical operations on the sinuses. An important point, I believe, is to be psychologically

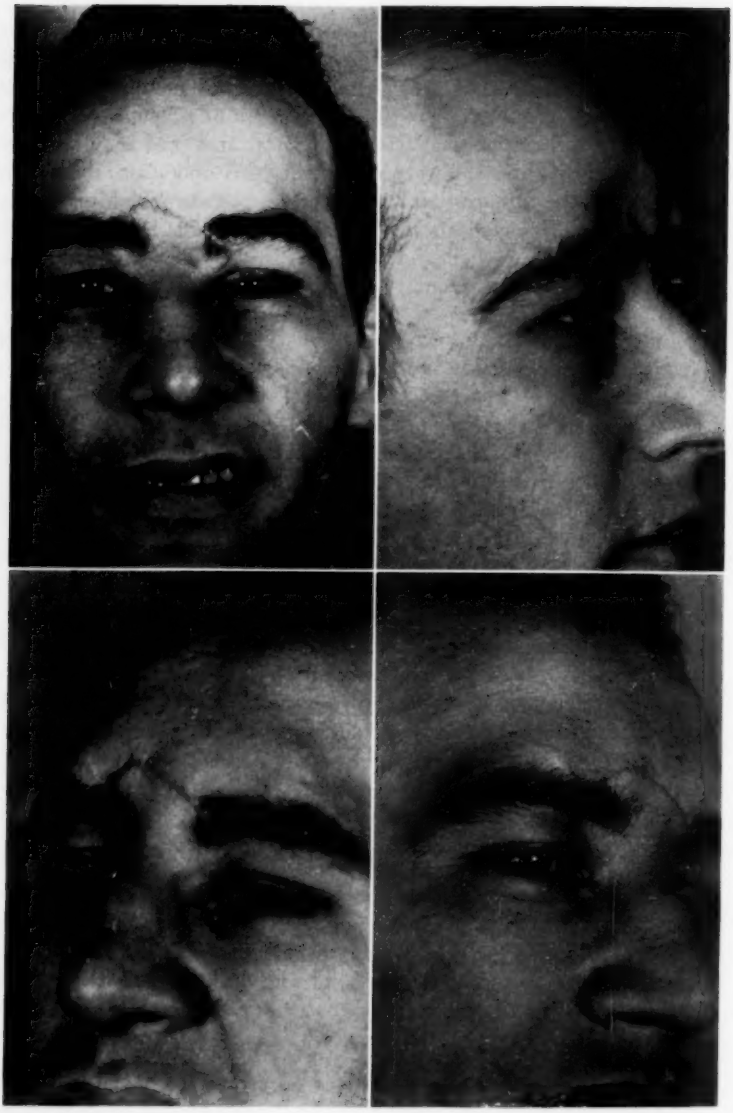


Fig. 1.—Case 1.—Above: Appearance before repair. Below: Appearance after repair by Dr. Bradford Cannon.

prepared to be thorough once we have committed ourselves to the obliterative operation.

When the obliterative operations were first performed, the ugly deformity which resulted deterred nearly all operators and patients, likewise, from these procedures. I think this point was over emphasized. Certainly there are many patients who have so suffered from the disease and from unsuccessful operations previously undertaken that the obliteration of the sinus, even with the deformity seems to have given such relief that they are satisfied with the result. To the surgeon it is not so agreeable to see a deformity that he has produced. But it is also a relief to know that the chances of the sinus becoming reinfected are reduced to a minimum.

Fortunately for those of us who do this operation today we have an excellent chance to overcome the deformity through the improvement in plastic repair of the defect by living autogenous bone grafts. I would like to give a few case histories which will illustrate the problems which I have encountered.

## REPORT OF CASES

CASE 1. (Figs. 1 and 2) A. S., a male, aged 20 years. The patient was seen on June 3, 1948, in consultation with Drs. Bradford Cannon, plastic surgeon, and Jost Mickelson, neurosurgeon, and Garrett Sullivan, ophthalmologist, at the Massachusetts General Hospital. The history related that on that day the patient was inflating a rubber tire on a wheel barrow when suddenly the tire blew up and struck him in the face and left hand. He was brought to the Baker Memorial of the Massachusetts General Hospital where an x-ray revealed a skull fracture involving the midfrontal region just above the frontal sinus, the posterior wall of the frontal, the right maxilla, the nasal bones, the ascending process of the maxillae and the lower orbital rim on the right, so that the alveolar process on this side was freely movable. There was a deep laceration of the upper lip. There was a cerebrospinal leak through a deep laceration in his forehead. The patient was conscious. On examination his right eye showed a severe laceration of the globe which eventually required removal of the eye. At this time the immediate problem was to debride the frontal sinus and examine the dura for a tear. Under intratracheal anesthesia a debridement of the frontal sinus was carried out by Dr. Mickelsen and myself. The laceration was so extensive that no further incision

Fig. 2.—Case 1.—X-ray showing large defect resulting from debridement of frontal bone fracture before bone graft repair.

was needed to uncover the entire front wall of the frontal bone. The anterior walls were fractured into many small fragments. On removing these the posterior wall appeared to be fractured also on the right side. This fracture line extended through the roof of the orbit on both sides, the roof of the right ethmoid and the cribriform plate including the crista galli. The cribrifrom and the crista gallie were freely movable although still attached to the frontal lobe dura. The glabella and nasal bones, likewise, were freely movable but attached to the overlying skin and periosteum. At this operation all the loose fragments were removed from the anterior and posterior walls from the roof of the orbit and the ethmoid on the right side including the adjacent cribriform plate. The crista galli and the remaining portion of the cribriform on the left side were left in place. A tear in the dura about one-half cm in diameter was found in the anterior cribriform area on the right side. This was closed with a silk suture which included a small piece of gelfoam thus stopping the cerebrospinal leak.

Following this the wound was closed and pressure was applied externally, thus obliterating the entire frontal sinus area. The fracture of the maxilla and the laceration of the lip were repaired and the patient was returned to bed in fair condition. Later the eye was removed at another operation. By September 24 the patient had sufficiently recovered from these procedures so that a plastic repair was done by Dr. Cannon using living bone from the iliac crest to fill in the defect of the frontal sinus. Since then the patient has done extremely well and has not had any infection resulting from the injury.

CASE 2. (Figs. 3 and 4) G. A., a male, aged 18 years. This case illustrates frontal sinusitis following a fracture of the skull with osteomyelitic complications. On March 8, 1951, I first saw the patient with the following history. During the previous October he had been struck on the head while at work. An x-ray at that time confirmed the presence of fracture of the left frontal sinus. The sinus then became infected and he was then treated with antibiotics. The infection appeared to subside. However, three days before I saw him he contracted a head cold. The sinus again became infected and on the night before admission to the Massachusetts Eye and Ear Infirmary he noted some swelling of the forehead and upper eyelids on the left side. He had a dull headache and a purulent discharge. The following day the swelling of the forehead had begun to subside but his eyelids were still edematous and inflamed. There was marked tenderness over this area. A preliminary incision and drainage of an orbital abscess was undertaken. On exposing the front face of the frontal sinus a large defect was discovered apparently in line with the fracture as shown by x-ray. There was pus within the sinus. Two days laters under intratracheal ether a radical obliterative frontal sinusectomy and ethmoidectomy was performed. A supraorbital incision was made to the outer limit of the frontal sinus and medially downward along the nasal bone to expose the lacrimal sac. The incision was carried upward medially and the periosteum was elevated to expose the entire front face of the left frontal sinus. The defect as before noted was found. There were two fracture lines on the front face of the frontal bone. Between them there was an area approximately 2 cm by 4 cm of soft osteomyelitic bone. Over the roof of the orbit there was another defect which represented a third fracture line. In the supraorbital rim there was a sharp spur which probably was due to a displaced fragment from the original trauma.

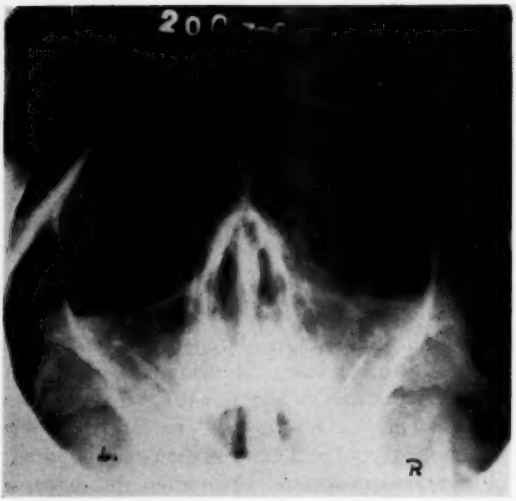



Fig. 3.—Case 2.—X-ray showing fracture of left frontal sinus

Fig. 4.—Case 2.—On left: Appearance before repair. On right: Excellent appearance after repair by Dr. Edgar M. Holmes.

In view of the osteomyelitis, a radical obliterative frontal operation was done. There was a large lobular extention of the left frontal sinus beyond the midline partly overlapping the right frontal sinus, due to an oblique deviation of the interfrontal septum. All the partial septa were removed and the entire mucosa covering the posterior wall was curretted away. Ethmoid extensions were found overlying the posterior portion of the orbit. A complete ethmoidectomy was done including exenteration of all the orbital extensions. A small area of soft bone in the lateral portion of the frontal sinus was removed exposing an area of normal dura. The sphenoid ostium was identified and a probe passed easily into the sinus which was normal. A few months later a plastic repair was done by Dr. Edgar M. Holmes with the excellent result as shown in the accompanying illustration. Again living bone from the iliac crest was used.

CASE 3. T. K., a male, aged 18. This patient was first seen by me, May 6, 1947, with the following story. He previously had had severe infection of his frontal sinus and under local treatment had failed to respond satisfactorily in spite of the usual bed rest, sulfadiazine, heat and frequent nasal shrinkage. Then he had part of his middle turbinate and many polyps removed. An exenteration of the anterior ethmoid cells was also done. He was then given penicillin intramuscularly and locally. He improved under this regime but about three weeks before I saw him his frontal sinus had again become reinfected. He had headache and no treatment locally at that time seemed to have very much effect on his condition. When I saw him he had x-ray evidence of pus in his right frontal sinus. There were polyps in his nose, and there was no drainage visible intranasally. Under local anesthesia an external right frontal-ethmoid and sphenoid operation was undertaken. At this operation the entire ethmoid appeared to have degenerated. There were no partitions visible, the entire labyrinth being composed of thick infected mucous membrane with an occasional polyp. There was pus in the frontal sinus and here the membrane was markedly thickened and slightly polypoid. He recovered from these procedures very well but there was a tendency for his nasal frontal duct to block off. For this reason tantalum foil was inserted to provide permanent drainage. This was done on July 3, 1947, and following this he got along quite well until September, when he caught another head cold and developed some edema and inflammation around his right eye. At this time he was at home and the doctor in charge removed the tantalum. Following

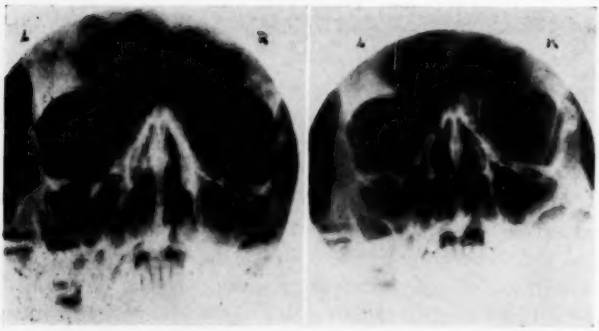


Fig. 5.—Case 6.—Right: X-ray of sinuses before latest operation. Many ethmoid cells can be seen over the roof of the frontal sinus. Large defect in posterior wall on the right is trephine for epidural abscess at a previous operation. Left: X-ray shows appearance after latest operation. The oval area in the roof of the orbit on the right represents the site of an ethmoid cell with unusually heavy bony outlines probably due to sclerosis.

this the nasofrontal passage closed off completely, and he returned to see me in October of the same year. Reoperation was done and pus was found just above the nasofrontal duct area. At this time as there appeared to be a low grade osteomyelitis of the front face of the frontal bone, an obliteration was undertaken. From this he recovered well. The large defect in the frontal area was repaired by Dr. Edgar M. Holmes, chief of the plastic service at the Massachusetts Eye and Ear Infirmary, the following autumn with very good results. From then till now, a period of eight years, he is symptom free.

Case 4. L. D., a male. I first saw this patient on December 17, 1945, for chronic right frontal sinusitis. Several years before that he had had a severe left frontal sinusitis with osteomyelitis. This was operated on by another rhinologist who did a partial obliteration of the left frontal sinus. When I saw him there was marked supraorbital tenderness on the right side. He had severe headache. X-ray showed mucoceles forming on the left and a very large mucocele on the right. There was marked thickening of the mucosa of the left antrum but without retained secretion. The ethmoids were clear. This time his right frontal sinus was the one chiefly involved. There-

fore, a right radical Lynch frontal operation was undertaken. This included his ethmoids and exposure of the sphenoid sinus as well. At this operation it was found that the interfrontal septum was gone, and there was a large opening from the upper part of the left frontal sinus across the midline into the right frontal sinus. Hoping that drainage could be established in this way into the right nostril, no other procedure was contemplated at this time. However, he did not do well. His nasofrontal duct blocked off and as his occupation required him to be exposed to very severe wintry weather, it seemed wiser to obliterate his sinus than to go on hoping that his sinuses could be kept open and that he could develop enough immunity to ward off further infection. Consequently, a reoperation was performed on February 4, 1949, and at this time, because the two sinuses were virtually connected, a bilateral radical obliterative procedure was undertaken. The entire floor of the right sinus was removed and on the left side what appeared to be a Killian bridge was discovered, which hid several mucoceles; therefore, this side was revised completely. The entire ethmoid region was revisualized and on this occasion every vestige of ethmoid extension was removed. The patient has had no further complications and no further inflammation or reinfection of his frontal area since then. In spite of the good results offered by plastic surgery, the patient has been content to let things stand as they are.

CASE 5. E. B., a female. This patient I saw first on February 14, 1950, with the following history. One year before she had had an acute fulminating left pansinusitis and was operated upon at another hospital. At that time she had had an obliterative frontal, ethmoid and a radical antrum. Unfortunately, during that operation her optic nerve was affected so that she had lost the vision on this side. When I saw her there was an exacerbation of infection on the left side with extension across the midline involving the right side. A revision of the left frontal sinus and an operation upon the right frontal sinus were undertaken. At this operation the left frontal sinus showed partial regeneration of the floor and remnants of the anterior wall. Pus was encountered in the midline near the interfrontal septum. There was more pathology than had appeared by x-ray. As part of the left frontal floor was still remaining, this was removed. This concealed some grossly infected mucous membrane. Following this a few posterior ethmoid cells were uncovered. A good portion of the middle turbinate appeared to be still present. All of

Fig. 6.—Case 7.—X-ray shows the Killian bridge with three areas of erosion by mucoceles. There are also many ethmoidal extensions over the roof of both frontal sinuses.

this was removed. The sphenoid sinus was visualized. It contained a little thick membrane. The antrum appeared to be grossly infected and on removing the ethmoid overhang, pus flowed from the left antrum into the nose in spite of the fact that there was a good intranasal lower meatus opening. The right frontal sinus was then exposed and the frontal wall and floor were removed. The mucous membrane appeared to be grossly infected in the region of the nasofrontal duct. The ethmoid was exenterated and the sphenoid sinus opening was visualized. Again the ethmoid overhang concealed gross infection in the antrum and, on removing it, pus came out freely from this side. Following this procedure the patient made a good recovery and has not had any recurrent infection in her frontal sinuses. Her antra have cleared up following intranasal window operations, adequate drainage having apparently taken care of them. This patient is quite content to let things stay as they are without a repair of the defect.

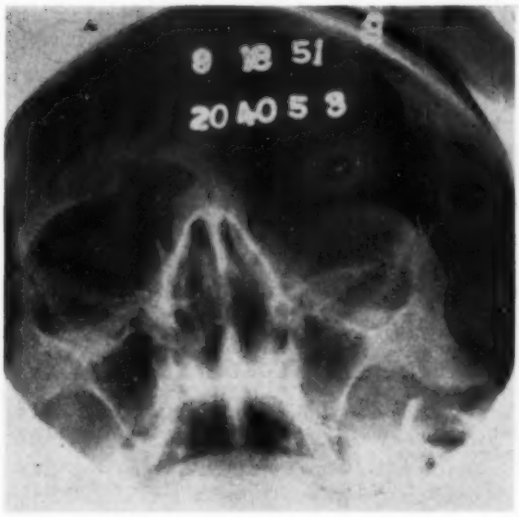



Fig. 7.—Case 8.—X-ray showing obliteration of right frontal sinus but with a large mucocele forming. This became infected and required reoperation.

CASE 6. (Fig. 5) A. G. The patient was first operated upon in 1936 and since then has had multiple operations to correct a chronic bilateral frontal sinusitis. The infection, however, had progressed so that when I first saw him, May 19, 1954, there had been multiple attempts at obliteration of both frontal sinuses. He had had a trephine of the posterior wall on the right side because of an epidural abscess. In spite of all this the patient had become reinfected from time to time and when I saw him there was another exacerbation. There was pus on both sides of his nose and marked tenderness and headache over the entire frontal area. At his request I operated on him on the Nose and Throat Service of the Massachusetts Eye and Ear Infirmary. A complete revision of the frontal area was done. Many mucoceles were discovered on both sides and cleaned out completely by curretting. Partial partitions were base-leveled still further. Then it appeared that ethmoidectomy had not been completed at previous operations. This was now undertaken. An aberrant cell

Fig. 8.—Case of Dr. Edgar M. Holmes.—Left: Appearance of deformity before repair. Right: Appearance after repair showing excellent result from use of autogenous bone graft.

Fig. 9.—Case of Dr. Edgar M. Holmes.—Left: Appearance at time of operation showing extent of defect. Right: Appearance after repair with autogenous bone graft. An excellent result.

was found in the nasal septum. This required removal of the portion of the nasal septum just behind the nasal bones. This presented a little problem as it was somewhat difficult to close the incisions in the midline at this point. However, this was successful and since then the patient has had no nasal discharge or crusting, no inflammation and the entire area seems to be in good condition. He has had very little upper respiratory infection and on each occasion no involvement of the frontal sinus region. Likewise, his general health and well-being have improved so that he now says that he feels better than he has for many years. To date he refuses plastic repair.

CASE 7. (Fig. 6) L. H., a male, aged 59 years. This patient was referred to me by Dr. Schall on April 30, 1954, having been sent to the Massachusetts Eye and Ear Infirmary by his own doctor in New Brunswick. This patient had had a severe frontal sinus infection twenty-five years before and had been operated upon in another city. At that time a bilateral Killian operation was performed, and many nasal polypi were removed then and on subsequent occasions. He was mildly diabetic which complicated the control of his infection. When I saw him he was not anxious to have another operation and had been specifically told by another nose and throat man that he should never be operated on again. With this prejudice to overcome, my first attempt was to explain to him the situation and to get his acute inflammation under control, the patient at that time having had another flare-up. This was done and two weeks later he reported back to me stating that if I desired to operate, he was willing to go through with it. This was done on May 18. The revision of the frontal sinuses on both sides was performed under general anesthesia. An incision was made from right to left across the supraorbital ridge. The periosteum was elevated from the bone both above and below the Killian bridge which was then removed with mallet and chisel and rongeurs. Large pyoceles were encountered over the roof of the orbit on both sides. All of these were removed. The entire mucous membrane was dissected away from these cells. The ethmoidectomy was then revised through the orbit. The wound was closed and a pressure dressing applied. The patient has done extremely well since then. His diabetes was kept under control and had improved as one would expect because of the elimination of focal infection. This patient also is quite content to let things stay as they are and not undertake plastic surgery, although undoubtedly it would make him look more presentable.

Fig. 10.—Case of Dr. Edgar M. Holmes.—Photograph showing excellent cosmetic result of autogenous bone graft repair.

Fig. 11.—Case of Dr. Edgar M. Holmes.—Photograph showing condition just prior to plastic repair. This was a case of ostectomy for osteomyelitis of the frontal bone.

CASE 8. (Fig. 7) J. B., a male, aged 67 years. I present this case merely to show what can happen even after a good obliterative operation of a frontal sinus in which, however, a little mucous membrane is missed and which then forms a large and bothersome mucocele. This shows in the accompanying x-ray. In short, the only difficulty in this case was the recurrence of the mucocele; the obliteration, otherwise, having been quite successful. Reoperation to remove the mucocele was all that was needed.

I am much obliged to Dr. Edgar M. Holmes and Dr. Bradford Cannon for permission to use photographs of some of their patients illustrating the postoperative results of plastic repair of the frontal sinus defect. In these cases autogenous bone grafts from the iliac crest were used, the bone being cut, shaved, and fitted to the defect at the time of the implant. Banked bone was not used and for this reason probably better results were obtained. The following photographs illustrate the preoperative and postoperative appearance of the patient.

## SUMMARY

A group of cases has been presented in whom simple operative procedures and the usual Lynch frontal operation do not seem to be indicated because of: 1) severe traumatic injury to the frontal sinus; 2) the presence of chronic osteomyelitic disease; 3) the history of repeated attempts at conservative operative measures which have resulted in the end in such complications that a revision of the frontal area by other than the most radical procedure would seem to give very little hope of success.

The operation under consideration entails removal of the anterior and inferior walls of the frontal sinus, the complete removal of the entire frontal sinus mucosa, the base leveling of all partial septa in sinus, a complete ethmoidectomy.

Previous failures at attempted obliteration have usually been the result of the presence of ethmoid cells, remnants of frontal mucosa and incomplete removal of the floor of the frontal sinus.

Once the operator has decided upon obliteration, he should not stop short of a complete and radical removal of these structures. The fear of disfigurement need no longer deter us as the defect can be successfully corrected by a living bone graft.

Eight case histories are given to illustrate the above points.

330 DARTMOUTH ST.

## REFERENCES

1. Kuhnt, Hermann: Über Die Entzündlichen Erkrankungen Der Stirnhöhlen Und Ihre Folgezustände. Wiesbaden, 1895.

2. Röpke: Die Radicaloperation bei chronischen Verschleimungen und Eiterungen der oberen Nasennebenhöhlen. Berlin, 1898.

3. Riedel-Schenke, Heinrich: Ueber die Stirnhöhlen und ihre Erkrankungen. Inaugural Dissertation, Jena, 1898.

4. Goodale, R. L.: Some Causes for Failure in Frontal Sinus Operations. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 51:3:648 (Sept.) 1942.

LIII

# UNILATERAL VOCAL CORD PARALYSIS

DANIEL S. CUNNING, M.D.

NEW YORK, N. Y.

Two years ago, I read with great interest an article[1] on unilateral vocal cord paralysis written by one of our distinguished colleagues, Doctor Louis H. Clerf. I thought at the time and am of the same opinion still that it was a great contribution to our laryngological literature. This is a subject that has annoyed me for many years.

I was so impressed with this article that I was determined to study our cases at the Manhattan Eye, Ear and Throat Hospital, adhering as closely as possible to Doctor Clerf's clear-cut and orderly presentation of the subject. Unfortunately, I have nothing new to offer but I felt it would be worthwhile to record our cases and compare them with Doctor Clerf's series.

One of the most unsatisfactory conditions we are called upon to treat is hoarseness due to unilateral vocal cord paralysis. The diagnosis is relatively easy to make but to give an accurate prognosis is most difficult unless we are able to determine the cause of the paralysis. These paresis are rarely caused by disease within the larynx itself and as Clerf says "it is not a distinct clinical entity but rather a symptom of some local manifestation." The most common cause is usually some involvement of the vagus or its recurrent laryngeal branch, somewhere between the jugular foramen and its entrance into the larynx proper.

Approximately 10 per cent are caused by some intracranial disease before the nerve leaves the skull such as tumor, abscess, bulbar paralysis or vascular accidents. When caused by a central lesion there is usually some paralysis of the muscles supplied by the glossopharyngeal and spinal accessory nerves, as their nuclei are so closely related in the brain. So the first thing we should do as soon as we note paralysis of a cord is to examine the pharynx, palate and tongue

---

From the Manhattan Eye, Ear and Throat Hospital.

to see if there is any paralysis of these organs and if so, we can feel quite sure that we are dealing with an intracranial lesion.

One interesting case develops this point: A 65 year old man, seen by Doctor Morley T. Smith at the New Rochelle Hospital, was perfectly well until four days prior to admission when he awakened early in the morning and began to cough up a thick mucus. He developed nausea and vomiting. This continued each day and the patient was unable to take solid food. His voice became hoarse and on the day of admission he spoke in a whisper. He became extremely fatigued and progressively lethargic. No similar episode had occurred in the past. Physical examination revealed paralysis of the left vocal cord, edema of the pyriform fossa with pooling of mucus in this area. There was also sluggishness of the pharyngeal reflexes and difficulty in swallowing. The patient was noted to have pulmonary edema and expired on the day of admission.

The clinical findings of nausea, vomiting, hoarseness, lethargy, paralysis of the left vocal cord and stupor followed by death were indicative of a lesion involving the pons. This lesion, usually an infarction, knocks out the olive as well as the fibers of the tenth nerve, the filaradicularia.

At autopsy, the brain was examined after formalin fixation for 48 hours. With multiple sections there was noted a gray nodule measuring 8 mm in diameter and 16 mm in length within the left side of the pons. This nodule had distorted the olive on the left side so much that its normal convoluted pattern was no longer recognizable. The lesion extended from the olive to within 8 mm of the floor of the fourth ventricle.

Sections through the brain failed to reveal any microscopic changes but for the lesion of the left side of the pons. This lesion was composed of an area of almost complete necrosis of the nerve fibers and nuclei. The lesion histologically consisted of a defect containing irregular strands of degenerated fibrils. The edge of the lesion was fairly well demarcated. Sections through the cervical cord failed to reveal any pathological changes. Sections through the intrinsic arteries of the brain failed to reveal a thrombus. Other portions of the pons were normal.

This lesion was an infarct which was well defined histologically and was undoubtedly on the basis of thrombosis of a left pontine branch of the basilar artery. The artery at this point was of micro-

scopic size and the thrombus could be easily missed histologically and grossly.

This interesting case shows the importance of a histologic study of the brain in these intracranial lesions.

The vast majority of unilateral cord paralysis (about 90 percent) are caused by peripheral lesions along the course of the vagus or recurrent laryngeal nerve. The left is more frequently involved than the right in the ratio of about ten to one and the reason is probably due to its longer and more tortuous course before reaching the larynx, although in this series the ratio was about two-and-one-half to one.

The position of the paralyzed cord and its tonicity should be noted in all cases whether it is in adduction, abduction or in the cadaveric position. King and Gregg[2] in 1948, showed by their meticulous and accurate anatomical dissections that 30 per cent of their recurrent laryngeal nerves divided extralaryngeally into two branches, the anterior branch supplying the adductor muscles and the posterior branch the abductor, and this is the reason why some of these paralyzed cords lie in adduction and some in abduction. If the recurrent nerve is injured before this division or if there is injury to the vagus itself, the cord will lie in the cadaveric position, that is, midway between abduction and adduction, completely paralyzed. Hinton and Weeks in their dissections found 76 per cent divided extralaryngeally. Morrison[3] found 43 per cent in his dissections of 200 recurrent nerves.

Rustard and Morrison who have done a marvelous piece of work on the anatomy of the recurrent laryngeal nerve, have shown that the nerve may be divided extralaryngeally in from one to six branches on one side with a single branch on the opposite. They are in full accord with Fowler and Hansen as to the location in which the nerve may be injured in thyroidectomy, that is, not at the lower pole as is usually assumed, but at the posterior lateral junction of the middle and lower thirds, just above the point where the chief branches of the inferior thyroid artery enter the gland and the nerve is often in intimate relation with the posterior surface of the gland.

### PARALYSIS OF THE VOCAL CORD OF PERIPHERAL ORIGIN

For the 15 years period from 1938 to 1953 inclusive 262 cases were studied in the Bronschoscopic Clinic of the Manhattan Eye, Ear

and Throat Hospital. 163 were males and 99 females. Eighty-two had involvement of the right cord, 180 had involvement of the left. The average age was 46. They were divided as follows:

| | | |
|---|---|---|
| Traumatic | 21 | |
| Mechanical | 29 | |
| Neoplastic | 72 | 27.5% |
| Toxic or Inflammatory | 58 | |
| Idiopathic | 82 | 31.3% |

### TOXIC AND INFLAMMATORY

Many infections and toxic agents seem to have an affinity for attacking the vagus or recurrent laryngeal nerve. There was but one case of cord paralysis following lead poisoning. This young man, 36 years of age, a chemical engineer, became hoarse quite suddenly overnight. He was seen two days later. Examination revealed an abductor paralysis of the left vocal cord. In the routine physical examination it was discovered that he had free lead in his blood and urine. Although he changed his position to one in the office of the company away from the laboratory where he was inhaling the fumes of molten lead, and his blood and urine became free of lead, his paralysis still persists after three years.

Many of the cases following acute infections such as acute upper respiratory infections, colds, virus infections, etc., regained mobility of the cord in two or three months. Three cases were apparently due to diabetes. In the routine medical check-up it was discovered that they were suffering from diabetes, with high blood sugars. They were all put on diet restrictions, insulin, etc. Two of the cases recovered mobility of the cord in two or three months. The third, although his diabetes was kept under control, did not recover (probably due to degeneration of the nerve). There were two cases of paralysis, with the cord in the cadaveric position, that were due to neuromas of the vagus nerve.

### MECHANICAL CAUSES

Under this heading are the cases of cord paralysis that are caused by pressure on the recurrent nerve itself, such as enlargement of the heart, apical tuberculosis, aortic aneurysm, in fact enlargement of any organ or gland that comes directly in contact with the nerve.

This can be readily explained when we remember the course of the nerve from the vagus, how it is in close proximity to the mediastinum, pleura, lungs, trachea and esophagus. In Clerf's series there were 15 cases of cord paralysis due to pulmonary tuberculosis and in all the 15 the left recurrent nerve was affected. In our series there were 11 due to tuberculosis with involvement of the left recurrent nerve, in six cases, and the right in five. In tuberculosis there are supposedly more patients with paralysis of the right recurrent than of the left. Why this should be is unknown as there are just as many patients with involvement of the left apex as there are the right.

### NEOPLASTIC CAUSES

As in Clerf's series, the most common cause for unilateral cord paralysis was neoplastic growths. There were 26 cases from carcinoma of the lung either by direct invasion of the nerve or metastasis, 17 from carcinoma of the esophagus, 11 from mediastinal metastasis, 12 from malignant nodes in the neck and 6 from cancer of the thyroid.

Out of the 262 cases studied, 72 were caused by maglignancies. One of those followed lymphosarcoma of the neck with an abductor paralysis. This case was treated by x-ray therapy and the cord returned to normal. It is now nine years since therapy. Mobility of the cord was regained three months after cessation of therapy.

In malignancies of the lung, mediastinum, esophagus and breast, paralysis of a cord is a bad omen and usually means that the disease is well advanced and as a rule is beyond hope and that surgery cannot eradicate the disease.

### TRAUMA

There were 21 cases following trauma; 17 followed surgery on the thyroid gland; three followed injuries to the neck from automobile accidents; one followed manipulation of the esophagus in the mediastium in an attempt to resect it for carcinoma. Paralysis of one or both cords following thyroid surgery is not uncommon and as a rule it is not due to cutting the nerve itself but to forgetting that in a large per cent of cases the nerve divides extralaryngeally into two or more branches and one or more of these branches are injured. Another common cause is injury to the branches of the inferior thyroid arteries. In clamping the vessels to control bleeding some of the branches of the nerve are included in the clamp pro-

ducing cord paralysis. It is therefore very important not to injure these vessels and cause bleeding.

It has been reported that paralysis of a cord can develop following child-birth either by pulling on the infant's neck or by improper application of the forceps.

It cannot be stressed too strongly that the larynx should be examined in every case before surgery is done on the thyroid so as to make sure that the cords are functioning normally.

In our attempt to arrive at the cause of these peripheral lesions a complete and thorough physical examination must be done by a competent internist, including the heart, lungs, abdomen, nervous system, blood chemistry, Wasserman, x-rays of the lungs, sinuses and neck. The blood and urine should also be examined for lead and arsenic. Bronchoscopy and esophagoscopy must also be done before the examination can be called complete. In spite of this thorough work-up, there were 82 cases or 31 per cent in which the cause of the paralysis could not be determined. Therefore it was impossible to give an accurate prognosis as to how long the hoarseness might persist.

Some of the patients have had their paralysis for years with no other signs or symptoms. Others recovered spontaneously with a normal functioning cord and a good voice, in all probability the cause being toxic or inflammatory in nature even though we were unable to find it.

## TREATMENT

When there is an abductor paralysis, the voice is usually good and no treatment is necessary except in cases where the voice is used more or less constantly as in school teachers, public speakers, singers, etc. Such patients often complain of marked fatigue when using their voices in their accustomed manner.

When there is an adductor paralysis, the unaffected cord is unable to cross the midline to meet its fellow on the opposite side and the voice is very poor. Morrison[4] has devised an operation for fixing the paralyzed cord in the midline. Such an operation has many technical difficulties and I have not dared to attempt it.

Most cases of cord paralysis should be placed in the hands of a competent voice instructor, as many times it is amazing how much they can be helped.

## CONCLUSIONS

1. Paralysis of the cord is not an uncommon cause of hoarseness.

2. In our series, an etiological factor was found in 68.7 per cent. In the remaining cases, 31.3 per cent, no cause could be found.

3. Every patient with an unilateral cord paralysis must be thoroughly studied—complete physical, neurological examination, x-ray and serologic studies.

4. The toxic and idiopathic groups made up more than half of the cases (53 per cent) in our series.

115 EAST 65TH STREET.

## REFERENCES

1. Clerf, L. H.: Unilateral Vocal Cord Paralysis, J. A. M. A. (Mar. 14) 1953.

2. King, B., and Gregg, R. L.: Anatomic Reasons for the Various Behaviors of Paralyzed Vocal Cords, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (Dec.) 1948.

3. Morrison, Lewis F.: Recurrent Laryngeal Nerve Paralysis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1952.

4. Morrison, Lewis F.: The Reverse King Operation, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 1948.

LIV

# GENERAL TONSILLECTOMY

## SUGGESTIONS FOR MANAGEMENT AND PERITONSILLAR INJECTION

PAUL M. MOORE, M.D.

CLEVELAND, OHIO

In glancing through the literature it would seem that the subject of tonsillectomy has been milked dry. It is hoped that it will be possible to express a few more drops with sufficient flavor to hold your interest. A good share of this presentation will be devoted to a condensation of many things that have been well established. A portion will deal with a procedure which has proved very satisfactory in the hands of the author. Lastly a few ideas will be offered as to how ACTH and cortisone may affect the operation for better or for worse.

There has been a considerable amount of comment concerning the emotional and psychological preparation of the child for surgery.[1-3] This phase of the problem is very important. Fortunately the majority of today's parents do an excellent job of telling the child why the operation is necessary and what he may expect. One mother called attention to a very clever record by Mercury Childcraft entitled "Peter Ponsil Lost His Tonsil." In this recording a little boy tells of his experiences in song and rhyme against a background of catchy music. This makes an interesting adventure of the whole experience and helps prepare the child for his operation. When one of the parents is nervous and fearful and transmits the fear to the child, there may be a real problem.

In our hospital the parents bring the child to the hospital the morning of the operation and stay with him until he is taken to surgery. They are with him through the rest of the day after he returns from the recovery room. He is thus shielded from the feeling of being abandoned among strangers. The operated cases are kept separated from those awaiting surgery. The ideal arrangement is the recovery room where the patients who have been operated upon remain until they have recovered from the effects of the anesthetic.

This not only keeps them from being seen by those awaiting operation, but insures careful supervision during the immediate postoperative period. Oxygen and suction are readily available. If there should be any bleeding or respiratory difficulty they are where they can receive immediate and expert attention.

There has been tremendous progress in considering the child's emotional reaction to surgery since the 1920's. At that time I observed the way tonsillectomies were done in a world-famous clinic in Europe. The nurse would sit down with the child in her lap, with its legs between hers. She then clamped a scissors hold on the child's legs and held the upper part of the child's body with one arm. A mask was then half filled with ether and the patient's face forced down over it. When the crying and struggling ceased, the patient was held upright while the surgeon inserted a mouth gag and operated as rapidly as possible without looking for bleeders. Even so, the child was beginning to struggle and cry by the time the adenoids were reached. Compare this with the consideration the little patient now receives. It is hard to imagine psychic trauma occurring when the child goes to sleep under the anesthetic while visiting with the anesthetist about pets, games or school.

The question of preanesthetic sedation of children has been raised by Eckenhoff.[4] He studied the effects of various combinations of atropine or scopolomine with pentobarbital and morphine on 1500 children. He stated that crying increased such hazards of anesthesia as cyanosis and respiratory depression. Scopolomine, pentobarbital and morphine reduced the incidence of crying but also produced more frequent respiratory depression and cyanosis. He felt that pentobarbital should be used but that morphine was contraindicated in children. We have used nothing but atropine in children under the age of 14. This is given in graded doses according to the age and size of the patient, as worked out by the department of anesthesia. One-half hour before operation the following doses are given:

| | |
|---|---|
| 0 - 2 years | no atropine |
| 2 - 3 years | 1/400 gr atropine |
| 3 - 4 years | 1/300 gr atropine |
| 5 - 12 years | 1/200 gr atropine |
| 13 - 20 years | 1/150 gr atropine |

At ages over 14 years they are given sodium pentobarbital 1½ to 3 gr and morphine 1/6 to ¼ gr. We prefer not to take chances with narcotic respiratory depression in children.

Some years ago I would approach an adult tonsillectomy under general anesthesia with a deep feeling of no enthusiasm. Added to the length of time required to anesthetize the patient was the excessive amount of bleeding usually encountered during the operation. The bleeding makes it difficult to keep the operation under adequate visual control. This in turn makes it almost impossible to hug the surgical capsule during the dissection and results in injury to the blood vessels lying in the muscle fibres just outside the capsule. More bleeding results and there are more vessels that require ligation. This is a marked contrast to the small amount of bleeding in the average local tonsillectomy in which the gag reflex has been controlled by the topical application of cocaine. Here the surgical anesthesia is produced by the injection of procaine with six minims of epinephrine to the ounce. The injected fluid distends the mucous membrane and enables the surgeon to stay close to the surgical capsule and so avoid the blood vessels just beyond. The epinephrine produces contraction of the blood vessels so that there is very little bleeding unless a vessel of some size is encountered. Very often no ligatures are required.

It was decided to apply the principle of peritonsillar injection to the tonsillectomies under general anesthesia to see if, 1) it would reduce the bleeding during the operation, and 2) if it would facilitate the dissection. The results have been most gratifying and this method has been used rountinely on children as well as adults for several years. The first trial, however, almost caused the idea to be abandoned. The surgical nurse misunderstood the directions. One tonsil was prepared by injecting around it and promptly turned dead white. When asked what solution she had provided, she said 1-1000 adrenalin. Needless to say, that tonsil was removed in record time. No harm resulted but several weeks passed before the procedure was tried again.

No originality is claimed for this method. Many of you may use it—at least in your adult cases. A careful search of the literature reveals only two papers mentioning peritonsillar injection in preparing adult general tonsillectomies. In both of these instances the injection was used for purposes of anesthesia. Blaisdell,[5] report-

ed the use of sodium pentothal and curare in 1949. He used local injection of procaine. He apparently did not use epinephrine, since he spoke of the excessive bleeding in his cases. Johnson,[6] in 1952, reported 189 cases of 14 years and older in whom he used pentothal sodium, curare, pontocaine and 1 per cent novocaine with six minims of 1-1000 adrenalin to the ounce. Usually less than 10 cc of the novocaine mixture were used for both sides. He used no intratracheal tube and encountered no postoperative laryngospasm. In 1944 Grant,[7] recorded a case of postoperative adenoid bleeding controlled by the injection of 20 cc of normal saline solution in the adenoid bed. Ball,[8] in 1948, advised the use of 1 per cent procaine with 30 minims of 1-1000 adrenalin chloride to the ounce as an injection to control postoperative tonsil and adenoid bleeding. If there have been any other reports of this procedure in the literature they have probably been buried in obscurity by the ambiguous nature of the titles selected for the articles. None have been found where it has been used routinely in children as well as adults.

## METHOD

The patient is placed with the head low and well crowned so that the blood will pool in the nasopharynx and will not run down toward the larynx. The Davis mouthgag with tongue blade attached is used. The adults are given sodium pentothal induction and intratracheal anesthesia with the intubation tube through the nose. The children receive vinyl ether induction and no intubation is used. Our anesthesiologists are all medical doctors and are experts at intratracheal anesthesia. They feel, as we do, that it is not necessary in the children and carries with it the added possibility of producing postoperative edema of the larynx in them. Seated at the head of the patient and using the Davis mouth gag the operator can easily inspect the larynx at any time by lifting up the epiglottis with the blunt dissector end of the pillar retractor.

Normal saline solution with six minims of 1-1000 epinephrine to the ounce has been selected for the injection. One per cent procaine with a similar amount of epinephrine was used for a time. This was abandoned for the normal saline because it was felt that the added anesthesia was unnecessary. The injection is carried out in the same manner as that which is used for the injection of procaine in the local tonsil operation. The injection is started at the upper pole just beneath the mucosa at its junction with the tonsil. This is made very superficially so that the membrane is ballooned out. The

needle is then pushed in deeply and the solution is injected around the upper pole of the tonsil. Injection is then made just beneath the mucous membrane of the posterior and anterior pillars. These injections are aided by pulling up the free margin of the soft palate with the pillar retractor, thus tensing these structures. An injection is then made just beneath the lower pole, and then thrusting the needle more deeply a second injection is made in the same place. From two to three cc of the solution is usually used on each side. Each tonsil is then grasped, pulled forward and up and 1.5 cc is injected through the anterior pillar lateral to the tonsil mass. In all the deeper injections the plunger should be pulled out to be sure that no blood vessel has been entered. After a moment's wait, to give the solution a chance to exert its action, the dissection is begun.

A variety of injections have been tried in the adenoid area. None of them has succeeded in reducing the amount of bleeding during their removal. Injection is now used only after their removal to help control any excessive bleeding from this area. The solution is injected and then pressure is brought to bear with a sponge. If this does not control the bleeding, a sponge, moistened with adrenalin and dusted with tannic acid powder, is applied and any visible blood vessels are ligated. In applying ligatures in either the adenoid or tonsil areas a slip knot is preferred to a needle ligature. Although a needle ligature will not slip off, the needle itself may penetrate a deeper vessel.

There has usually been very little bleeding during the removal of the tonsils, even in the adult cases. This has been sufficiently obvious for the anesthesiologists, who handle the suction as well as the anesthesia, to remark about it on many occasions. We had thought of measuring the blood lost during this part of the operation and comparing it with that lost in cases done without the peritonsillar injection. We were deterred by the report of King,[9] who measured the blood loss in 176 adenotonsillectomies in children of 2 to 11 years of age. He was investigating the effect of Vitamin K on the blood lost at operation. In the Vitamin K group the blood lost ranged from a maximum of 150 cc to a minimum of 20 cc. In the control group it ranged from 165 cc to 14 cc.

## CONTRAINDICATIONS

The solution used for the peritonsillar injection consists of normal saline to which has been added six minims of 1-1000 epinephrine

to the ounce. Usually 10 cc or less is used in the preparation of the tonsils for dissection. Thus only two minims of 1-1000 epinephrine is used. Another minim may be used in helping control the adenoid bleeding. In spite of this small amount of epinephrine, it is felt that it should not be used in certain situations. In these cases normal saline alone is injected.

1. Epinephrine is not used in children under the age of two years. Neither do they receive any atropine. It is felt that it is unwise to use any drugs other than antibiotics at this age.

2. Epinephrine increases the blood sugar. It will therefore increase the need for insulin in diabetic children. It should not be used in these cases.

3. Hosler[10] sounds a word of caution, especially regarding the use of epinephrine in coronary sclerosis. To quote—"An irritable heart or one under the influence of an anesthetic may be the seat of sudden precipitation of ventricular fibrillation by epinephrine." He states that this is due to the drug causing the general rate of oxidation of all tissues to increase and therefore creating a greater need for oxygen.

4. Hamilton[11] advises against the use of epinephrine with cyclopropane. He states that cyclopropane not uncommonly produces cardiac arrhythmias and that epinephrine further enhances this tendency. The epinephrine-normal saline mixture has been used on one adult patient under cyclopropane anesthesia without any untoward results.

## DISCUSSION OF CASES

Although this method was instituted in 1943 only the last 312 cases have been surveyed for this study. In this group there were 183 patients, aged three years through six years. The patients under the age of three years numbered seventeen and those over fourteen numberd twenty-eight. More adults are asking for general anesthesia. With the improvement in the technique of anesthesia and the use of the peritonsillar injection these patients are being granted their wish.

No figures concerning the incidence of primary bleeding were found in the literature. The question whether injection of normal saline with epinephrine will increase the number of primary hemorrhages may be raised. There is no reason to suppose that this would

occur more frequently in the general tonsillectomies than it would in the local operation where the same injection is used with procaine and epinephrine. There were six cases of primary hemorrhage, or 1.923 per cent, although no patient was allowed to leave the operating table until both tonsillar fossae and the adenoid bed were dry. Three of these cases were bleeding from the adenoid bed and three from a tonsillar fossa. The tonsillar bleeding was felt to be the result of ligatures slipping off during the recovery from the anesthetic.

The last ten years have produced many careful and excellent investigations into the causes of secondary or late bleeding in adenotonsillectomies.[12-23] The great majority of these have incriminated acetylsalicylic acid. In 1934 Ersner, Myers and Ersner[24] conducted extensive experiments on the effect of saliva on the clotting of blood. They found that saliva reduced the clotting time from 10 to 15 minutes down to 2 to 3 minutes. Acidification of the saliva prevented it from exerting this clotting action. Acetylsalicylic acid used locally in the mouth in post-tonsillectomy cases is changed by the saliva into free salicylic acid. This acid tends to digest and loosen the coagulum in the fossae prematurely.[14] Bleeding results and the normal coagulating effect of the saliva is prevented by its acidification. In 1945 Singer[23] called attention to the lack of late bleeding in tonsillectomies in Central Europe where they did not use acetylsalicylic acid as compared with the United States where it was used freely. Neivert,[19] in 1945, stated that acetylsalicylic acid lowered the blood prothrombin and thus tended to produce bleeding from the wound. Livingstone and Neary[18] disagreed with this.

The present series has been divided into two groups. In the earlier group, comprising 89 cases, the local use of acetylsalicylic acid was not only allowed but advised. In this group there were six instances of secondary bleeding. Two of these were from the adenoid area. This represents a percentage of 6.741. In the second group of 223 cases the local use of acetylsalicylic acid was discouraged and more recently prohibited. In this group there were only three instances of secondary bleeding, or 1.345 per cent.

Penicillin is used in selected cases. Vitamin K is given both pre- and postoperatively as a routine measure.

### EFOCAINE

Recently the literature has contained a number of reports on the use of a longacting local anesthetic, efocaine, to control post-

operative pain in various parts of the body. Penn[25] and Allen[26] reported good results in relieving post-tonsillectomy pain. They both warn against injecting the drug superficially. The fossae must be completely dry before the injection is made. Allen states that injection in the region of the upper pole produces edema of the uvula and soft palate. Davidson, Boles and Snyderman[27] injected only one side in each patient. The differential in pain between the two sides was so variable that they concluded that the drug was of questionable value.

Marks[28] encountered such a marked edema of the uvula and soft palate in one patient that he considered the possibility of having to do a tracheotomy. He reported good relief of pain but encountered large pale sloughs at the sites of the injections by the seventh postoperative day. Half of his cases had marked secondary hemorrhages.

An ominous note of warning is given by Angerer, Su and Head.[29] Their patient was suffering from a bilateral pulmonary tuberculosis. A thorocoplasty had been done on the left side. There was encapsulated fluid in the pleural cavity on the right side. The pain on the right side was severe and efocaine was injected in the right eighth intercostal space in the posterior axillary line. This injection was made without preliminary aspiration. The injection was made slowly. After 1.5 cc had been injected the patient suddenly complained of severe, electric, knife-like pains in the back and radiating down both legs. Both legs were soon paralyzed and the patient collapsed. Death occurred 44 hours later. A transverse myelitis of the spinal cord, thrombophlebitis of the intercostal vein and thrombosis of the intercostal artery were found at autopsy.

It is to be noted that this case was published after the reports of Penn, Allen, Davidson et al and Marks. The tonsillar fossae are as near the spinal cord as the posterior axillary line. Is post-tonsillectomy pain of sufficient magnitude to justify such tremendous risks in seeking its alleviation?

### ACTH AND CORTISONE

The systemic use of ACTH and cortisone has far reaching effects on all of the metabolic processes of the body. Some of these effects may have an influence on the operation of tonsillectomy.

Kinney[30] encountered excessive bleeding during local tonsillectomy operations on two patients who had been on cortisone for ocular infections. He had extensive blood studies made on the second patient preoperatively, because of his experience with the first. The blood counts, coagulation and bleeding times, platelet counts and prothrombin values all were within normal limits.

The general concensus of opinion is that cortisone does not produce a decrease in the clotting mechanism.

On the other hand, Margulis[31] found an increase in the circulating heparin levels of 0.04 or more and in the coagulation time of three minutes or more in patients subjected to surgical stress, systemic cortisone or ACTH therapy. These increased values appeared within the first twenty-four hours and then returned to normal or even below normal. Following this there was another slight but steady rise with a peak between the third and fifth days. The elevated levels persisted for three to four days after withdrawal of the drug.

Brust, Ransohoff and Reiser[32] reported an increase in the blood pressure and the pulse pressure in patients receiving ACTH. After three weeks two of their patients developed swollen pink fingertips which persisted until the drug was stopped. They stated "A mere prick for blood counts produced an unusual gush of blood."

In planning a tonsillectomy on a patient receiving ACTH or cortisone it might be well to withdraw the drug four or five days before the operation is done.

Fortunately the question of severe surgical shock seldom enters the problem of tonsillectomy, yet deaths do occur on the operating table during the course of this operation. When they occur, they come with devastating swiftness. Many of these are due to anoxia. These can be avoided by an alert anesthetist and surgeon working as a team and constantly watching the airway and providing adequate oxygenation. Although the thymicolymphatic diathesis has been greatly discredited during the last few years, autopsy frequently shows these patients to have enlarged thymus, enlarged spleen and generalized hyperplasia of the lymphoid tissues of the body. These individuals seem to be peculiarly sensitive and may develop severe shock from relatively minor trauma. Stephen[33] says " . . . the pale, fat, listless, 'thymic' child can be anesthetized safely, if careful attention is given to the depth of narcosis and degree of oxygenation.

It is believed the patient suspected of having 'status lymphaticus' has less resistance than the normal child, and requires a smaller concentration of the anesthetic agent in his blood stream to provide satisfactory operating conditions."

When this condition has been recognized preoperatively, it has been the custom to give the patient roentgen irradiation over the thymus gland. This was done to reduce the size of the gland and to provide some protection against any untoward reactions. Selye and Jacot[34] state that cortisone has a thymolytic effect which is greatly enhanced by the simultaneous treatment with ACTH. Conn[35] suggests the use of 100-200 mgs of cortisone or hydrocortisone for two days preoperatively if the condition is recognized before surgery. It would seem that this might offer better protection than the roentgen therapy against the reaction to stress in these individuals.

Recently ACTH, cortisone and hydrocortisone have been reported to have a very beneficial effect on hypotension due to surgical shock. Hume and Moore[36] cite a patient with carcinoma of the anus who required extensive surgery. She developed shock during the first operation. There was hypotension, oliguria and peripheral circulatory collapse. She rallied from this under treatment with blood transfusions, vasopressors and other routine measures. Fifteen minutes after the start of the second operation the blood pressure fell to zero, although there was no undue blood loss. Stimulants and blood transfusions were given. The blood pressure rose to 50/40. She continued hypotensive all day. She was placed on ACTH, 30 mgm every six hours and the blood pressure rose further to 120/80. She was continued on ACTH and later cortisone. Ten days later the third operation was done without any hypotension developing, although the blood loss was much greater than in either of the other two operations.

Hydrocortisone has a much greater potency than cortisone. It can now be given intravenously. In 1954 Rukes, Orr and Forsham[37] reported its use in four cases of surgical shock. They added to this series in a report to the New York Academy of Sciences this year. Two of the four previously reported cases developed shock with hypotension during the course of extensive operative procedures. The first patient had been on a course of cortisone for 28 days followed by ACTH for five days. The operation was done six days later. Hypotension developed in spite of two transfusions and Vasoxyl®

intravenously. When the pressure reached 60/45, a slow intravenous drip of 100 mgm of hydrocortisone in 1000 cc of 5 per cent dextrose was started. Five minutes later the blood pressure rose to 100/70 and 25 minutes later it reached 130/80. The other three patients showed similar prompt response to the intravenous use of hydrocortisone in 5 per cent dextrose. They stated that 93 per cent of the hydrocortisone given intravenously is excreted within the next 24 hours. Blood loss should be replaced by transfusions and vasopressors should be used. The hydrocortisone potentiates the action of the vasopressors.

Conn[35] also suggests the intravenous use of hydrocortisone in patients who develop severe hypotension during or following operations. He recommends the drip method, using 100 mgm of hydrocortisone in 5 per cent glucose solution. If the shock is so extreme and precipitous as to produce a cardiac stand-still, immediate cardiac resuscitation seems to offer the only hope.

## CONCLUSIONS

1. The general management of tonsillectomies done under general anesthesia has been reviewed.

2. A method of peritonsillar injection has been described which reduces the amount of bleeding during the operation and aids in the dissection.

3. There is a considerable amount of hazard in the use of efocaine to relieve post-tonsillectomy pain.

4. Treatment with cortisone over a period of time may lead to excessive bleeding during a tonsil operation.

5. A short period of preoperative treatment with cortisone or hydrocortisone in the "thymicolymphatic" child might help protect him from the hazards of surgical trauma.

6. The intravenous use of hydrocortisone has been found to be useful in combating the hypotension of surgical shock.

10515 Carnegie Ave.

## REFERENCES

1. Levy, David M.: Psychic Trauma of Operations in Children, Amer. J. Dis. of Children 69:7-26 (Jan.) 1945.

2. Coleman, Lester L.: The Psychologic Implications of Tonsillectomy New York State J. of Med. 50:1225-1228 (May 15) 1950.

3. Coleman, Lester L.: The Emotional Preparation of Children for Surgery, Transactions Amer. Laryng. Rhinolog. and Otolog. Soc., 1954. Television Kinescope shown at meeting in May, 1954.

4. Eckenhoff, James E.: Preanesthetic Sedation of Children, Arch. Otolaryngol. 57:411-416 (Apr.) 1953.

5. Blaisdell, Irl H.: The Use of Sodium Pentothal Anesthesia in Adult Tonsillectomies, Laryngoscope 59:721-725 (July) 1949.

6. Johnson, Fordyce: Adult Tonsillectomies Using Pentothal Sodium, Curare, Pontocaine and Novocaine as the Anesthetic, Laryngoscope 62:704-708 (July) 1952.

7. Grant, Heman: Hydrostatic Pressure in Adenoid Hemorrhage, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 53:576-577 (Sept.) 1944.

8. Ball, Chas. E.: Nasal and Postoperative Tonsil and Adenoid Hemorrhage, Tex. State J. of Med. 44:437-440 (Oct.) 1948.

9. King, James T.: Effect of Vitamin K Compound (Synkavite) on Computed Blood Loss During Adenotonsillectomy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 63:1029-1030 (Dec.) 1954.

10. Hosler, Robert M.: Cardiac Arrest from the Otolaryngologist's Viewpoint, Arch. Otolaryngol. 57:371-377 (Apr.) 1953.

11. Hamilton, Norval E.: General Anesthesia in Otolaryngology, Trans. Amer. Laryngol. Rhinolog. and Otolog. Soc., pp. 263-268, 1954.

12. Coombes, Arthur J.: A Study of Delayed Bleeding Following Removal of Tonsils and Adenoids, Laryngoscope 60:296-307 (Mar.) 1950.

13. Fox, Samuel L. and West, A. Brooks, Jr.: Vitamin K and Late Tonsillar Bleeding, Bull. School of Med. Univ. of Maryland 32:215-220 (Apr.) 1948.

14. Fox, Samuel L.: Bleeding Following Tonsillectomy, Laryngoscope 62:414-425 (Apr.) 1952.

15. James, A. G.: Hemorrhage Following Tonsillectomy, Related to Weather During a Twelve Year Period, Ann. West. Med. and Surg. 2:558-561 (Dec.) 1948.

16. Jones, E. H.: The Prevention of Secondary Hemorrhage Following Tonsillectomy, South. M. J. 42:124-127 (Feb.) 1949.

17. Jones, E. H.: Better Tonsillectomies and Adenoidectomies; Fewer Postoperative Hemorrhages, South M. J. 16:15-20 (Jan.) 1953.

18. Livingstone, G. S. and Neary, E. R.: The Question of Prothrombinopenic Hemorrhage From Post-tonsillectomy Use of Chewing Gum Containing Acetylsalicylic Acid, Arch. Otolaryngol. 47:1-6 (Jan.) 1948.

19. Neivert, H.: Late Secondary Tonsillar Hemorrhage. I. Studies of Prothrombin and Vitamin K, Arch. Otolaryngol. 42:12-18 (July) 1945.

20. Neivert, H., Pirk, Leo A. and Engleberg, Richard: Late Secondary Tonsillar Hemorrhage. II Studies of Ascorbic Acid. Arch. Otolaryng. 43:568-577 (June) 1946.

21. Neivert, H.: Acetylsalicylic Acid and Bleeding from the Tonsillar Wound, Arch. Otolaryngol. 47:524-526 (Apr.) 1948.

22. Pearlman, S. J. and Loewy, A.: Secondary Tonsillar and Adenoid Hemorrhage in Relation to Barometric Pressure Changes, Arch. Otolaryngol. 53:527-529 (May) 1951.

23. Singer, Rudolf: Acetylsalicylic Acid. A Probable Cause for Secondary Post-tonsillectomy Hemorrhage, Arch. Otolaryngol. 42:19-20 (July) 1945.

24. Ersner, Mathew S., Myers, David and Ersner, Wm.: A Clinical and Experimental Study of the Action of Saliva on Blood Coagulation and Wound Healing in Surgery of the Oral Cavity and Throat, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 43:114-146 (Mar.) 1934.

25. Penn, S. E.: Control of Post-tonsillectomy Pain, Arch. Otolaryng. 56:59-60 (July) 1952.

26. Allen, Risden T.: New Method for Relieving Postoperative Pain Following Tonsillectomy, Arch. Otolaryngol. 57:86 (Jan.) 1953.

27. Davidson, Morris, Boles, Robert G. and Synderman, Sanford C.: A Prolonged Anesthetic in Control of Post-tonsillectomy Pain, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 61:1046-1047 (Dec.) 1952.

28. Marks, Roland F.: The Effect of Efocaine in the Control of Postoperative Pain, Laryngoscope 63:861-862 (Sept.) 1953.

29. Angerer, Alden L., Su, Hung H. and Head, Jerome R.: Death Following Use of Efocaine, J. A. M. A. 153:550-552 (Oct. 10) 1953.

30. Kinney, Charles E.: Discussion of Present Concepts of the Activities of the Pituitary-Adrenal Mechanism and of the Adrenal Steroids, by J. W. Conn Middle Sect, Meet. Amer. Otol. Rhinol. and Laryngol. Soc. (Jan.) 1955.

31. Margulis, R. R.: Blood Coagulation as Affected by ACTH and Surgical Stress, Surg. Forum Amer. Col. Surg. 507-515 (Oct.) 1950.

32. Brust, Albert A., Ransohoff, Wm. and Reiser, Morton F.: Vascular Responses to ACTH and Alterations in Sodium Intake, Proc. Second Clin. ACTH Conf., Blakiston Co., 1951, 1:177-195.

33. Stephen, C. R.: Operating Room Fatalities in Pediatric Surgery, Anes. and Analg. 31:36-44 (Jan.-Feb.) 1952.

34. Selye, Hans and Jacot, Bernard: On the Effect of Various Steroids upon the Extra-adrenal Thymolytic Action of ACTH Acta Endocrinol. 9:333-336 (Apr.) 1952.

35. Conn, J. W.: Personal Communication.

36. Hume, D. M. and Moore, F. D.: The Use of ACTH, Cortisone and Adrenal Cortical Extracts in Surgical Patients, Proc. Sec. Clin. ACTH Conf., Blakiston Co., 1951, 2:289-303.

37. Rukes, J. Max, Orr, Richard H. and Forsham, Peter H.: Clinical Uses of Intravenous Hydrocortisone, Metabolism 3:481-488 (Nov.) 1954.

* * *

*(Scientific papers of the American Laryngological Association to be continued.)*

# The Scientific Papers of the American Otological Society

## LV

## AUDITORY FATIGUE AND ADAPTATION IN THE DIFFERENTIAL DIAGNOSIS OF END-ORGAN DISEASE

J. D. Hood

London, England

For the otologist the subject of hearing tests is one of very great practical importance. From hearing tests he seeks the means to measure his patients' deafness and, more than this, to identify the locus and nature of the deafening disease.

When, therefore, we hear of new additions to our knowledge of the scientific aspects of hearing, it is sometimes right to enquire how these help the practical otologist to solve his problems. I shall, therefore, endeavour to lay stress on the more practical implications of some of the new knowledge of hearing and its functional investigation that workers in this field have put at our disposal in the course of the last decade. From this we can select, I am glad to say, a number of items of clear-cut value to the practical otologist, among them the loudness recruitment test of Fowler, which has now taken its place as a valuable clinical adjunct in the differential diagnosis of lesions of the cochlear nerve, and of the end-organ of hearing.

The value of this test arises from the finding[1] that loudness recruitment, though constantly present in disease of the end-organ, is characteristically absent in lesions of the VIII nerve, a finding which has been confirmed by many writers.[2-5,7]

Now allied to the phenomenon of loudness recruitment we have certain others, which have in the past been grouped together with a certain lack of discrimination under the heading of auditory fatigue.

From the Otological Research Unit, Medical Research Council, National Hospital, Queen Square, London.

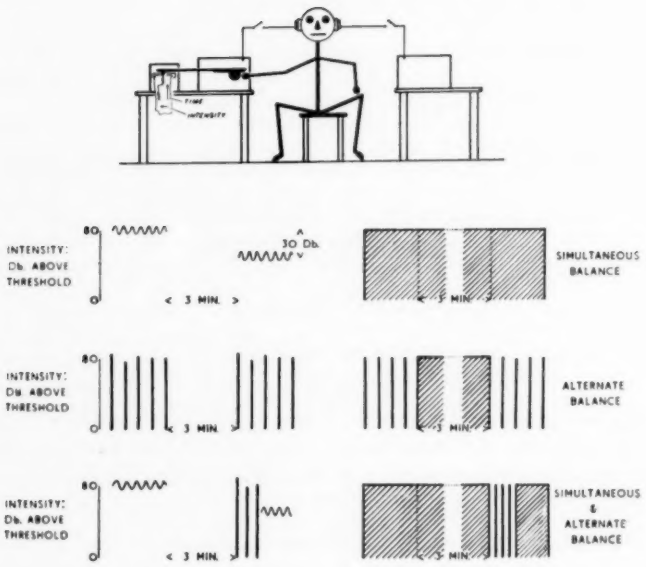



Fig. 1

While a certain overlapping of the functional effects of these phenomena sometimes makes their separation difficult, it is possible nevertheless to recognize three quite distinct physiological entities: adaptation, true physiological fatigue and, finally, trauma.

Since it is impossible in the course of this short communication to deal comprehensively with all three, I propose to restrict my remarks for the most part to the phenomenon of adaptation.

I should like to begin by reminding you briefly of the manner in which adaptation manifests itself in the normal subject. This is shown in Figure 1. Two audiometers supply the two receivers worn on the left and right ears of the subject. The frequency is the same in both receivers, and the intensity in the left ear is fixed at, say 80 db. The intensity at the right ear is continuously variable, and a simple pen recorder accurately records the intensity variations in this ear.

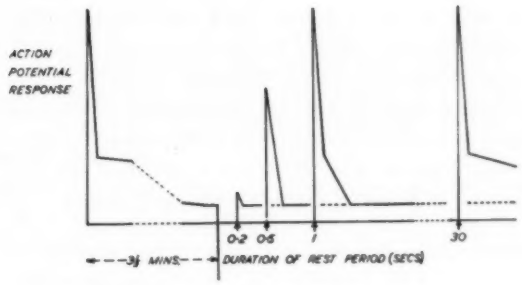



Fig. 2

Let us begin with the first of the three test procedures shown in this diagram.

Initially the sounds are applied simultaneously, and a loudness balance is made by varying the intensity at the right ear. This is attained with equal intensities, 80 db, in both ears. The tone in the right ear is then removed for three minutes, and that in the left ear is sustained throughout this period. The variable tone is then reapplied to the right ear, and a further loudness balance is made. It will be seen that this is now attained with an intensity of only 50 db. In other words, during the three minute period of application of the tone to the left ear a considerable loss of sensitivity has occurred, which is measured by the change, 30 db in the level at the right ear, required to give a loudness balance.

This, however, is only one aspect of this remarkable phenomenon. The other and equally important characteristic is shown in the second of these test procedures. In this, the initial loudness balance has been made by applying short pulses of sound alternately to the two ears. Once again, loudness balance is attained with equal intensities at the two ears. This is followed, as before, with a sustained stimulation of the left ear for three minutes, the right ear being rested.

Following this a final alternate loudness balance is made, as before, and you will see that there is now no change whatsoever in the intensities at the two ears. In fact, no adaptation is demonstrable.

At first sight, therefore, it would seem that the application of the interrupted tone to the left ear, following the sustained tone, had abolished the adaptation which we know must have occurred. That this is not so is shown by the third test procedure.

In this you will see that we begin as in Procedure 1 with a simultaneous loudness balance, followed by a sustained tone in the left ear. At this point, however, the procedure is varied. The sustained tone is followed first by a brief period of alternate loudness balancing and finally by a simultaneous balance. You will see that, as in Procedure 2, no sensitivity loss due to adaptation is revealed by the alternate balancing here. It is, however, at once revealed when the simultaneous balance is resumed.

We may say, therefore, that the response from the left ear, following the application of the sustained tone, is one of complete normality to short interrupted tones, but relapses once more to its adapted level if the sustained tone is reapplied.

In order to understand the striking difference in the result of these two test procedures, it is necessary to refer to the electrophysiological studies of Matthews upon the action potential responses arising from single muscle stretch receptors. Matthews' results are summarized diagrammatically in Figure 2.

When a stimulus is applied to such an end-organ, the action potential response consists of an initial high frequency discharge known as the on-effect. The duration of this initial burst of impulses is brief, being of the order of 0.2 seconds, and is followed by a slow decline in the discharge frequency with time. This decline is termed "adaptation."

If, following adaptation, the stimulus is removed and reapplied after a rest period greater than about one second, the on-effect is present and indeed undiminished, but continued stimulation results in rapid relapse of the response, that is to say, an accelerated adaptation to a level which is determined by the duration of the rest period.

We have termed these two phenomena on-effect normality and relapse, and it is pertinent to consider here their bearing upon the subjective cochlear responses I have just described.

Let us consider these again. In Procedure 1 of Figure 1 we see clearly revealed the cochlear counterpart of the essential component of adaptation, namely sensitivity loss due to sustained stimulation.

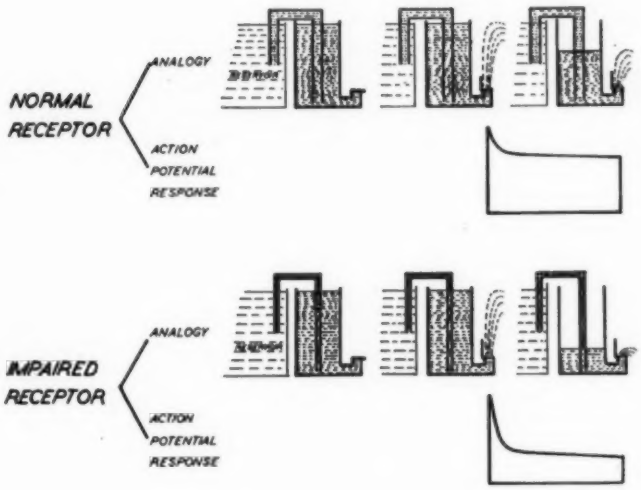



Fig. 3

In Procedure 2 we see the cochlear counterpart of on-effect normality. That is to say, the adapted end-organ is capable of initiating on-effects of normal magnitude.

Finally, in Procedure 3, we see the cochlear counterpart of relapse. That is to say, following the application of the sustained stimulus the response to brief interrupted stimuli is normal but relapses at once to the adapted level on reapplication of the sustained stimulus.

It is expedient at this point to consider how these phenomena, derived from the study of the normal end-organ, can be related to the diseased end-organ. Now adaptation may be described as the attainment of a state of equilibrium between the energy which is expended in the response, and the energy which becomes available to maintain it. The capacity of a receptor to respond to a sustained stimulus is dictated, therefore, by the rate at which fresh energy can be supplied to it.

Now it is not unreasonable to expect that in end-organ disease the energy reserve will be impaired, due possibly to ionic changes in the surrounding cochlear fluids, resulting in a subnormal equilibrial balance between input and output of energy. I will show presently that there is abundant evidence for this point of view. For the time being, however, I should like to direct your attention to an important similarity between the normal adapted end-organ and the diseased end-organ. Both exhibit this phenomenon of on-effect normality, and with sustained stimulation both rapidly relapse to a low response level. In the case of the diseased end-organ, the relapse phenomenon may be markedly accentuated, and this we have termed "pathological relapse."

The essential features of adaptation in the normal and impaired receptor expressed in terms of a simple analogy are shown in Figure 3.

The upper set of tanks represents a normal receptor; the lower set an impaired receptor. The capacity of each is the same, but the supply pipe to the lower tank is of much smaller diameter than that to the upper tank. The first tank in each case represents the quiescent condition of the two receptors, and it will be seen that they are identical, i.e., the height of the water level in each is the same.

When the taps are opened, corresponding to the response of the respective receptor to a stimulus, the initial fountains are of equal height as in the case of the two on-effects. In time, the water level in the two tanks falls to a level at which a state of equilibrium exists between the in-flow and the out-flow of the water. This is the condition of the adapted receptor when the energy supplied just balances the energy dissipated. Since the supply to the lower tank is reduced, the water level at equilibrium is low and the height of the fountain much reduced, corresponding to the adapted level of the action potential response of the impaired receptor. In other words, the energy reserve is deficient.

I have already referred to the phenomenon of loudness recruitment, and its occurrence in diseases of the end organ, and you are probably all familiar with the manner of its investigation in unilateral deafness by means of the alternate binaural balancing technique.

The results of such a procedure in two typical cases of end-organ deafness are shown plotted on conventional diagrams in the upper

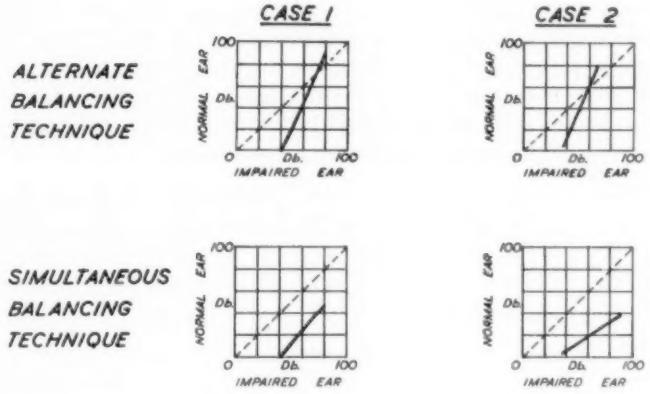



Fig. 4

half of Figure 4. The vertical and horizontal scales of the graphs refer to sound intensities applied respectively to the normal and affected ears of the subjects. The dotted lines passing through the origin are carried through a series of intensity points which give equal sensations of loudness in the two ears of a normal subject. Since the hearing is equal in the two ears the angle of the line is 45 degrees.

The full line curves show the occurrence of loudness recruitment in the two subjects in each of whom the deafness was due to unilateral Ménière's disease. It will be seen that in one, equal loudness is attained with an intensity of 70 db in both ears, and in the other with an intensity of 60 db in both ears. In both, over-recruitment is present.

Now let us consider the result obtained in the same two subjects with a balancing procedure whereby the sounds are applied simultaneously to both ears. In this the tones are continuous and are not interrupted at any time. The two lower curves show the results obtained and it will be seen that they are markedly different from the upper curves. In one subject loudness recruitment has been abolished altogether and in the other reversed.

These results would appear to be the direct subjective manifestations of the two phenomena to which I have earlier referred, on-effect

normality and pathological relapse. In the alternate binaural loudness balancing procedure, short tone pulses are used giving rise to normal on-effects and loudness recruitment when the stimulus level is high, whereas in the simultaneous balancing procedure the sustained tones used give rise to the condition of pathological relapse with a considerable decline in sensitivity resulting, as I have shown, in the abolition or reversal of loudness recruitment.

These factors must be taken into account in the design of test procedures for the investigation of the loudness recruitment phenomenon. This means that the duration and spacing of the interval between successive stimuli to one ear should be so arranged as to involve only on-effects.

Within these limits the phenomenon can as well be investigated by the alternate binaural balancing procedure as by a difference limen technique. But any technique which involves sustained stimulation is likely to bring into play the pathological adaptation of the sustained response, which as I have shown involves the elimination or even reversal of recruitment.

Let us turn now to the consideration of the occurrence of adaptation at threshold intensities. At this point it is pertinent to recall my previous statement that adaptation, pathological or otherwise, is the attainment of a state of equilibrium between the energy which is expended in the response, and the energy which becomes available to maintain it. In this respect therefore, it is, unlike true physiological fatigue, independent of the intensity of the stimulus and for this reason it is as likely to occur at intensities which evoke the faintest sensations, as those which evoke the loudest.

Its presence may be demonstrated in a number of ways and I propose finally to give you a description of two procedures which have been found to be of some value in the clinical differentiation of end-organ disease. The first of these is a simple test which reveals the presence of pathological adaptation or relapse in a striking manner. The results from three subjects with Ménière's disease at a frequency of 2000 cycles are shown in Figure 5. The first point on each graph represents the threshold of hearing obtained in the conventional manner with an interrupted tone. After this was determined, the intensity of the tone was raised by 5 db and applied continuously, the subject being asked to say when it could no longer be heard. In the normal subject, or one with conductive deafness,

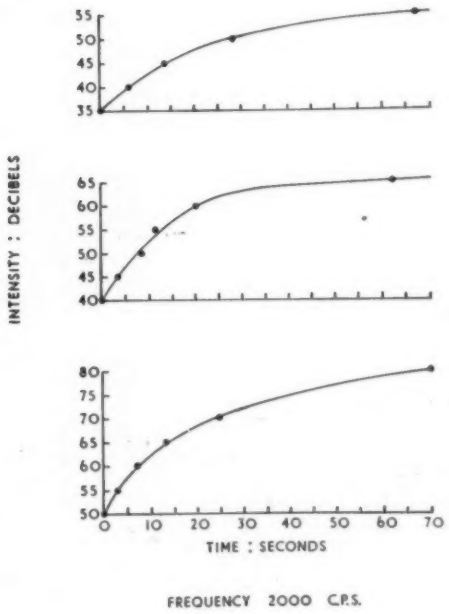



Fig. 5

an elevation of 5 db above threshold would result in the persistence of tonal sensation indefinitely. In these subjects with end-organ disease, however, you will observe that after only some five or six seconds of sustained stimulation, the sensation of tone had disappeared completely. After a rest period of one minute or more the procedure was repeated with the intensity of the tone raised successively by 5 db at each test. You will see that with higher tonal intensities the persistence of sensation becomes progressively longer until eventually an intensity is reached which evokes a sensation of tone indefinitely. This final intensity however, is some 20 or 30 db above the threshold intensity and this is the measure of the magnitude of the loss of sensitivity due to pathological adaptation.

There are two reasons why this loss of response must be attributed to adaptation and not to fatigue.

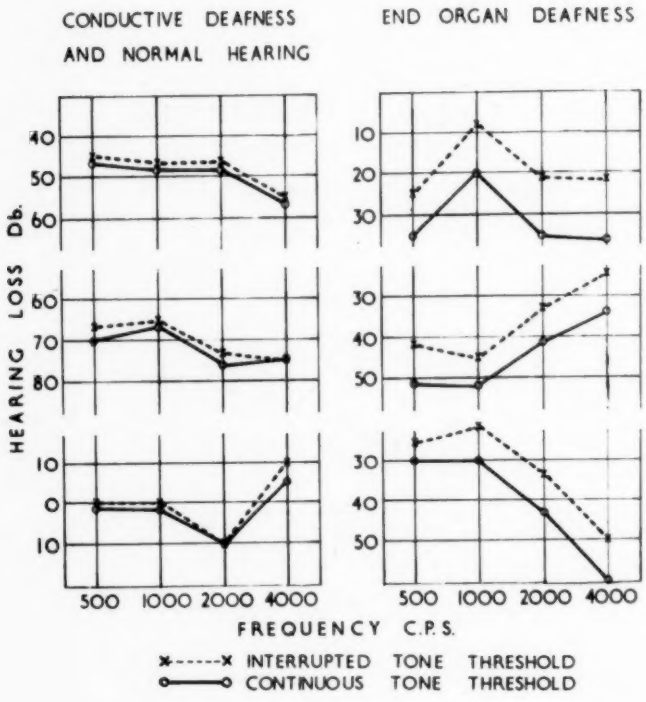



Fig. 6

In the first place, if the stimulus is continued to a point at which it is no longer perceived and is then interrupted and at once reapplied, then an immediate restoration of sensitivity occurs, and the tone is once more heard. If from this point the stimulus is again sustained then it falls more rapidly than before to an inaudible level.

In other words, the reapplication of the tone produced an on-effect with immediate restoration of loudness. Following this, however, the energy reserve of the receptors being already exhausted, their response rapidly relapses to their adapted state with the consequent extinction of sensation.

The second reason is perhaps an obvious one to those familiar with the investigations of Davis and his co-workers and more recently Ruedi in their studies of the rather different phenomena of auditory fatigue. These authors measured fatigue as an elevation of the threshold of hearing following previous intense stimulation. There is common agreement that fatigue is of persistent nature and that recovery therefrom is slow.

In both these respects, therefore, the threshold elevation described here, is markedly different from fatigue. It has resulted, as I have shown, not from any intense stimulation, but solely from the sustained responses of the receptors concerned to stimuli of exceedingly low intensities. Furthermore, this threshold elevation does not persist.

A further example of the ease with which the phenomena can be revealed, is provided by the results of a further series of tests in which we have used two different stimulus forms. First we have determined the threshold using a pure tone audiometer which has a tone interrupter switch with a rapid build-up of the order of 30 milli-seconds.

The almost square wave pulses which result are considered likely, therefore, to evoke the maximal on-effects. Secondly we have determined the threshold by a different technique, in which the intensity of the tone was gradually increased from a subliminal level at a rate of 2 db per second and the intensity noted when the tone was first heard.

The audiograms obtained from one normal and two subjects with conductive deafness using these two techniques are shown on the left of Figure 6. The lower full line curves are the thresholds obtained with continuous tones in the manner described. The upper dotted curves are the thresholds obtained with the interrupted tone and it will be seen that in general the difference between the two is slight, being of the order of 2 or 3 db.

On the right are shown the same tests applied in three subjects with Ménière's disease. It will be seen that the differences in the results of the two techniques are very striking and considerably greater than those obtained with normal subjects.

It would seem that the pathological adaptation or relapse which we have shown to occur in these subjects with end-organ disease takes

place with such rapidity that no on-effect at all is initiated when the intensity is increased gradually in the manner described. Instead, relapse occurs during the increase in the intensity of the tone and the response of the end-organs has reached a low level before the tone is heard.

In contrast, the short interrupted stimuli produce on-effects of high magnitude giving rise to a much lower threshold.

In conclusion, it seems only necessary to add that the test procedures described are by no means unique in the demonstration which they provide of adaptation in the diseased end-organ. Others based upon these principles could readily be devised and would no doubt be equally effective.

My aim has been two-fold. First to stress the importance of the physiological principles underlying the phenomena of adaptation both in the design of all audiometric tests of cochlear function, and in the interpretation of the results. Secondly to show how the use of tests, designed upon the basis of these principles enables us to effect the important clinical diagnosis of deafness due to disease of the cochlear end-organs. The tests that I have described have already proved to be of some value in this respect and there is every indication that clinical audiometery will benefit from further investigations in this interesting field of study.

NATIONAL HOSPITAL.

REFERENCES

1. Dix, M. R., Hallpike, C. S., and Hood, J. D.: Observations Upon the Loudness Recruitment Phenomenon, With Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve. Proc. R. Soc. Med. 41:516, 1948.

2. Ely, G., and Williams, H. L.: Recruitment of Loudness in the Differential Diagnosis of End Organ and Nerve Fibre Deafness. Laryngoscope 61:400, 1951.

3. Kristensen, H. K., and Jepson, O.: Recruitment in Otoneurological Diagnostics. Acta Oto-Laryng. 42:553, 1952.

4. Lundborg, J.: Diagnostic Problems Concerning Acoustic Tumours. Acta Oto-Laryng., Suppl. XCIX, 1952.

5. Lüscher, E.: The Difference Limen of Intensity Variations of Pure Tones and Its Diagnostic Significance. Proc. R. Soc. Med. 43:1116, 1950.

6. Matthews, B. H. C.: The Response of a Single End Organ. J. Physiol. 71:64, 1931.

7. Schuknecht, H. F., and Woellner, R. C.: An Experimental and Clinical Study of Deafness from Lesions of the Cochlear Nerve. J. Laryng. 69:75, 1955.

LVI

# AN ADDITIONAL FIVE YEAR REVIEW OF SOME CASES OF VERTIGO REPORTED IN 1949

W. J. McNally, M.D.

AND

E. A. Stuart, M.D.

Montreal, Canada

A review of 200 cases of vertigo was presented by McNally, Stuart, McKercher and Lockhart[10] as part of a symposium on vertigo at the IVth International Congress of Otolaryngology, held in London, July, 1949. The selection of these cases was almost consecutive. The sexes were equally represented and the ages at onset ranged from 10 to 79 years. Many of the cases had had their symptoms for five years or more prior to that report.

In the final analysis 78 out of the 200 cases were classed as True Ménière's Syndrome (Labyrinthine Hydrops). Important findings were that 47 of the cases had evidence of cardiovascular disease and 29 showed organic disease of the central nervous system. Spontaneous remissions of vertigo were common and averaged about four years in length with or without treatment. Because of the long remissions it was decided that five years would be required as the minimum period free of vertigo before a patient could be classed as "cured" regardless of whether or not the patient was receiving treatment. The term "temporary relief" was used to indicate freedom from vertigo for a period of at least six months, but for less than five years. The immediate relief which may follow the use of any medications was called "symptomatic relief."

It was thought that it might be interesting to review these 200 cases in 1954, five years later. It was hoped in so far as possible to ascertain what had been the medical course of each patient and how subsequent developments affected the diagnoses. Since only 27 of

From the Department of Otolaryngology, Royal Victoria Hospital and from the Montreal Neurological Institute.

these patients have reported for otological re-examination in the past five years, we have had to depend for our information on the results of a questionnaire followed up by innumerable letters and telephone calls. When possible and pertinent, the patient's physician was contacted for details of general health. We have traced 195 of the 200 patients.

It was hoped that we might make some assessment of the effect of treatment in the various classifications. It soon became apparent that the details of treatments followed, which could be obtained from the patients through a questionnaire, are so difficult to interpret in relation to dosage, frequency of administration, relation to symptomatology, etc., that the attempt was abandoned.

It was decided that it would be best to find out about the presence or absence of vertigo, tinnitus and deafness in the various classes and the relationship of these symptoms to serious organic diseases. The Results columns in Table I refer to the presence or absence of vertigo.

During the intervening five years a number of significant contributions have appeared in the literature and only those which are relevant to this series will be discussed. For a complete review of the literature see Williams.[13] Dix, Hallpike and Hood[4] clarified the differential diagnosis of Ménière's syndrome. These authors described two types of perceptive deafness: End-organ disease, essentially a disease of the hair cells in Corti's organ, is typified by Ménière's syndrome (Labyrinthine Hydrops): Nerve-fibre disease is typified by VIIIth nerve tumour. They reported that the binaural loudness recruitment test of Fowler, which had previously been considered diagnostic of perceptive deafness generally, is usually present in cases of labyrinthine end-organ disease but is not found in cases of nerve-fibre disease. When examined by speech audiometry, cases of end-organ disease show a lowering of discrimination for speech at higher intensities and a lowered threshold of discomfort. Cases of nerve-fibre disease may have good discrimination for speech and a high threshold of discomfort. These findings have since been confirmed by Eby and Williams,[5] Reger and Kos,[12] and by others. Only three unoperated cases of true Ménière's syndrome with unilateral hearing loss have reported for re-examination and all three showed recruitment. The report of Dix et al[3] brought about a revival of interest in the recruitment phenomenon. The difference limen test[9] which

deals with the determination of variations in the estimation of threshold difference is thought to be analogous to the recruitment phenomenon.

Hood[7] stated that when difference limen tests are carried out on cases with end-organ disease, using continuous threshold stimulation, the results may indicate a certain degree of pathological adaptation, a slow decline in discharge frequency, rather than be an indication of the recruitment phenomenon. He stated further that when there is end-organ disease the initial response to tones of short duration may approximate to or exceed the normal if the stimulus level is high, but that this response is not sustained if the stimulus is continued.

Because the interpretation of the difference limen test is still open to question, we shall not discuss the matter further.

Dix and Hallpike[3] suggested a reclassification of those cases of vertigo which are frequently grouped as Atypical or Pseudo-Ménière's Syndrome. Under Vestibular Neuronitis they included those cases which had paroxysmal vertigo, without tinnitus or deafness. The results of the vestibular tests were abnormal in most of these cases. The galvanic response was reduced in some cases. In many there was a history of previous febrile illness or evidence of a focus of infection. They concluded that the lesion in such a case was probably central to Scarpa's ganglion because of the reduction of response to the galvanic stimulation. They considered the lesion a benign one and reported good results following elimination of the focus of infection. Under Positional Nystagmus they grouped 100 cases which manifested positional nystagmus corresponding to Nylén's Direction-fixed, Type II. They stressed two characteristics in this group: that the nystagmus was essentially paroxysmal and that the course of the disease was benign. The cochlear and vestibular functions were usually found to be normal but there was evidence of middle ear disease in many. They reported the post mortem findings in one patient who died with a glioma involving the basal ganglia and the upper part of the brain stem on the left side. There was degeneration of the spiral ganglia in the right cochlea; the right utricle and saccule showed absence of otolithic membranes, disorganization of the sensory cells and thickening of the subepithelial connective tissues with irregular cellular infiltration. They concluded that the positional nystagmus in this patient was due to disease in

the right utricle, but how could they exclude the possibility that it was from disease of the right saccule? It may have been caused by the disease involving the basal ganglia and the upper part of the brain stem.

Nylén[11] discussed the subject of positional nystagmus and stated that positional nystagmus which occurs in various disturbances of the inner ear may occur, and possibly with greater frequency, when the lesion is in the central parts of the vestibular system.

Lindsay[8] makes the statement that postural vertigo and its accompanying positional nystagmus is the most common type of vestibular disturbance in diseases of the central nervous system. It occurs less frequently in peripheral ear disease.

As part of a symposium on Ménière's disease held by the section of Otology of the Royal Society of Medicine, London, 1954, Cawthorne and Hewlett[1] reported a series of 1902 cases of vertigo. Sixty-one per cent were due to Ménière's disease. Labyrinthectomy was carried out in 288 cases. It was reported that in six per cent of the patients, the other ear subsequently became affected.

Day had previously described an operation on the labyrinth for Ménière's disease in which coagulation was used with the idea of effecting a partial destruction of the internal ear in the hope that the cochlea might be spared. In 1952 Day[2] revised his technique and recommended a complete ablation of the internal ear as described by Cawthorne.

Dandy reported the results of VIII nerve section on a series of 401 cases of Ménière's disease in 1941. A follow-up on this series of cases was reported by Green and Douglas in 1951.[6] Vertigo was relieved in 90 per cent of the whole series, whereas only 37 per cent of the cases were relieved of tinnitus. Twenty-three cases had been submitted to differential nerve section in an attempt to spare hearing. In this small group vertigo was relieved in 56 per cent and tinnitus in 27 per cent. It was noted that after partial nerve section the hearing slowly declined in 48 per cent. Facial paralysis which occurred after nerve section was temporary in 25 per cent and was permanent in 10 per cent.

When our study was begun in 1949, the cases were arbitrarily divided into two groups—those with the complete triad (115 cases) being grouped under True Ménière's Syndrome and those in which

the triad was incomplete under Atypical Ménière's Syndrome. Before the investigation was completed, the cases under the heading of True Ménière's Syndrome were reassessed and only those cases in which the history and the signs and symptoms were compatible with an intralabyrinthine hydrops were retained. Cases in which such a diagnosis could not be established but which did not have definite evidence of other organic disease were placed under the classification of Atypical Ménière's Syndrome. This latter was a group of patients in which it was thought that other symptoms might develop at a later date which would ultimately clarify the diagnosis. It was further thought that many of these patients would probably develop the symptomatology of True Ménière's Syndrome. In the present review —five years later, it has been a great surprise to find that not one of the 35 cases grouped as Atypical Ménière's Syndrome has developed signs that would justify its being reclassified as a case of True Ménière's Syndrome. However, fourteen have been regrouped because they have developed signs diagnostic of other conditions; for instance, five now show signs of cardiovascular disease (see Table I). On the other hand, the group of cases of True Ménière's Syndrome has been reduced from 78 to 55 patients. Eight of these have been classified as Atypical Ménière's Syndrome because the original and subsequent evidence of intralabyrinthine disease was judged to be insufficient.

### TRUE MÉNIÈRE'S SYNDROME

Of the 78 cases originally diagnosed as True Ménière's Syndrome, 24 have been reclassified. All have been traced.

Three cases have been changed to Hypertension. Case 14 had had suggestive evidence of hypertension in 1949 and now at 66 years of age still has vertigo with definite signs of cardiac decompensation. Case 28, aged 75 years, has been free from vertigo for four years; the blood pressure is 180/100 and there is a family history of hypertension. Case 68, now aged 66 years, is free from vertigo. The blood pressure which was 160/110 in 1949 has risen to 210/110.

Two cases have been changed to Cerebrovascular Disease. In Case 181 the symptoms were not relieved by VIII nerve section in 1948. The patient developed signs of progressive cerebral deterioration and death occurred in 1952 at the age of 67 years. Case 186, aged 57 years, has persistent vertigo and has developed attacks of unconsciousness suggestive of cerebral circulatory changes.

Case 70, aged 42, has been reclassified as Otosclerosis because the hearing was improved by a fenestration operation done elsewhere in 1950. There is still mild vertigo.

Two cases have been reclassified as Psychosomatic. Case 49 has been under psychiatric treatment. Case 199 has been regarded by the physician as a case of anxiety neurosis.

Eight cases have been reclassified as Atypical Ménière's Syndrome. Case 17 had been erroneously classified as True Ménière's Syndrome in 1949. Originally there was no hearing loss and no tinnitus and none has developed in the past five years in spite of persisting vertigo. Case 34 had tinnitus; the hearing has remained normal; sphenopalatine ganglion neuralgia occurred prior to 1949 and now there is tingling in hands and feet; momentary attacks of vertigo occur but only when the patient is erect. Case 35 had a slight loss of hearing which has not progressed and there has been no tinnitus in the past five years in spite of persistent vertigo. Case 60 has never had tinnitus, has had a bilateral hearing loss which was confined to 8000 cycles in 1944, and has not changed. The patient has not had vertigo in the past five years. In Case 84 the vertigo began when the patient was aged 67, and ceased ten years ago at the age of 80; tinnitus has not developed. Case 106, free from vertigo for five years, had a hearing loss in childhood; was exposed to noisy industry and has had suppurative otitis media, all of which might explain a hearing loss which has not progressed in the past five years. Case 149 has slight unilateral hearing loss and tinnitus; there was pancreatitis and a cholecystectomy was performed in 1929; a breast removal was performed and irradiation given for carcinoma in 1949. Emotional instability and a labile blood pressure are thought to account for loss of consciousness and vertiginous attacks. Case 168 was thought to be an incipient case of Ménière's syndrome but a hearing loss has never developed in spite of persistence of vertigo. Central nervous system disease has been suspected but never established.

Eight cases were reclassified as Toxic Labyrinthitis. Case 8 had slight unilateral hearing loss, hypo-active labyrinth, and tinnitus on the same side and was extremely introspective. Vertigo had lessened following antral drainage in 1941. Recurrence of vertigo was associated with allergic manifestations. Case 40 was suffering from extreme fatigue before the onset of tinnitus and vertigo; there was no hearing loss and since having relinquished some responsibilities

the patient has been symptom-free. Case 43 had a unilateral hearing loss consisting of a drop at 8000 cycles and this loss has been progressive; infected tonsils were removed in 1948; a partial gastrectomy was done in 1954; there is a history of gall bladder disease. Vertigo and tinnitus persist. Case 57 had tinnitus and a hearing loss at 8000 cycles in one ear and this may have been due to gun fire to which the patient attributed the symptoms; infected tonsils were removed prior to 1949; there has been no recurrence of vertigo for 10 years. In Case 61 the hearing is normal; there still is tinnitus and positional vertigo without nystagmus. The basal metabolic rate was minus 22 per cent in 1954. The patient is subject to fatigue and anxiety. Case 162 had very little hearing loss which has not increased; tinnitus has ceased. The vertigo recurs during periods of excessive fatigue. Case 183 has had a unilateral hearing loss which has not progressed. The tinnitus was transient. Each of two attacks of vertigo followed upper respiratory infection. Case 190 has no hearing loss; is extremely nervous. The patient claims that tinnitus and vertigo have lessened with improvement in general health.

*Addition.* There has been only one addition to the group of True Ménière's Syndrome. Case 5 was classed as Psychosomatic in the 1949 survey. The original diagnosis had been True Ménière's Syndrome and a labyrinthine ablation had been performed in 1946 but the patient continued to have vertigo and was extremely neurotic. The gall bladder was removed in 1946; a nephropexy was performed in 1947 and a hysterectomy in 1948. There has been no vertigo in the past five years and the case has been reclassified as one of True Ménière's Syndrome. One might argue that this case should be classified as Toxic Labyrinthitis.

There were 54 of the original cases in the group of True Ménière's Syndrome in which the information obtained about their behaviour during the past five years supports the diagnosis of labyrinthine hydrops. It is interesting to note from Table I that 16 are classed as Cured; 22 as Temporary Relief; 4 as Symptomatic Relief and 10 as No Relief. There were no deaths in this group after 1949. During this last five year period only two cases were operated upon, and both had a labyrinthine ablation, Case 187 in 1952 and Case 175 in 1954.

True Ménière's Syndrome now contains 55 cases. This number represents 27.5 per cent of the 200 cases of vertigo in this series. This

is in striking contrast to the figures of Cawthorne who in 1954 reported the presence of Ménière's disease in 61 per cent of 1902 cases of vertigo.

### ATYPICAL MÉNIÈRE'S SYNDROME

Thirty-five cases were classed as Atypical Ménière's Syndrome in 1949. One patient who is employed as a secretary in foreign service has not provided us with detailed information. Fourteen cases have been reclassified.

Two cases without deafness, that had shown definite evidence of neurological disease in 1949 and who have not in the past five years developed signs which would allow of a definite diagnosis being made, have been transferred to a group designated as Neurological—Lesion Undiagnosed. Case 20 has had no tinnitus but has had headaches with diplopia and elevated spinal fluid protein. Case 126 has had indefinite tinnitus in 1947 and has peripheral neuritis and absent labyrinthine reactions.

Three cases have been reclassified as Psychosomatic. All three have normal hearing and two are known to have normal caloric reactions; they all have psychoneurotic episodes. One of the three cases (23) has had no tinnitus and is free from vertigo. Case 37 has had no tinnitus and has daily attacks of vertigo. Case 59 has had transient unilateral tinnitus and has vertigo when under mental strain.

Two patients (51 and 109) have developed signs of hypertension and have accordingly been reclassified. They both have slight and indefinite attacks of vertigo. The hearing in each case has remained normal. Their present ages are 54 and 56 years. One had no tinnitus and the other transient tinnitus. Vertigo preceded the evidence of hypertension by about 10 and 12 years.

Three cases have developed coronary thrombosis. Case 99, now 75 years of age, had a single attack of vertigo and tinnitus with normal hearing in 1946. Coronary thrombosis developed in 1949; there was no recurrence of the vertigo. Case 133 developed coronary thrombosis in 1954 at the age of 63 years; a single attack of vertigo occurred in 1944 without tinnitus or deafness; there has been no recurrence. Case 167 had the first coronary attack in 1949. There was a single attack of vertigo without tinnitus or deafness in 1947. Death from coronary disease occurred in 1954 at the age of 55 years.

There were four cases reclassified as Toxic Labyrinthitis. In Case 3 vertigo unaccompanied by tinnitus or deafness but with positional nystagmus has been associated with fatigue; there was a history of excessive use of alcohol. Misuse of alcohol was discontinued and infected tonsils were removed in 1948. Since then the vertigo has been less severe. Case 22 continues to have tinnitus and vertigo associated with severe mental strain and overwork; hearing loss is minimal. Case 63 has had no tinnitus and no deafness. A cholecystectomy was performed in 1950 since when the vertigo has occurred less frequently. Case 169 who has not had tinnitus or deafness but who did have positional nystagmus has suffered from extreme fatigue and anorexia of unknown origin; there has been freedom from vertigo for five years.

*Additions.* Eleven cases have been transferred to the group of Atypical Ménière's Syndrome; 8 from the True Ménière's Syndrome group; 2 from Hypertension and 1 from the Cerebral Atrophy group. The reasons for these transfers are stated in the case descriptions of the respective groups. This group of Atypical Ménière's Syndrome now contains 31 patients.

In this group of Atypical Ménière's Syndrome 20 of the original cases have been retained because in the past five years they have not developed signs which were specifically diagnostic. The hearing remained normal in 16 of the 20 cases. Three of the remaining cases which had hearing loss had no tinnitus—and the twentieth case had a hearing loss and tinnitus but also had loss of consciousness without other diagnostic signs of central nervous system disease.

One patient (Case 150) who has been retained in this group died of pneumonia at the age of 90 years in 1954.

The results in this group are as follows: 10 Cured; 6 Temporary Relief; 3 Symptomatic Relief; and 11 No Relief.

### TOXIC LABYRINTHITIS

Four of the original five patients in this group have been traced and all four have been cured of vertigo. Two cases (52 and 129) both of which had tinnitus and one a unilateral deafness, ceased to have vertigo after removal of a focus of infection, in each case the gall bladder. Two cases (46 and 147) both had suffered from vertigo while extremely fatigued and overworked; both had normal hearing

and one had tinnitus; relief from vertigo followed improvement in their general health.

*Additions.* Twelve cases have been added to this group, 8 from True Ménière's Syndrome and 4 from Atypical Ménière's Syndrome and they have been discussed under these headings.

### HYPERTENSION

There were originally 25 cases in this group. All have been traced.

Four cases have been transferred from the group. Two cases developed definite signs of coronary thrombosis. Case 56, aged 71 and retired from business, had a coronary attack in 1950; prior to 1949 he had had vertigo with loss of consciousness; there has been no recurrence of the vertigo. Case 3 has been unable to work for five years because of repeated attacks of coronary thrombosis but the vertigo has been infrequent. Two cases (130 and 139) have been reclassified as Atypical Ménière's Syndrome because the borderline blood pressure elevations reported in 1949 have not worsened in subsequent examinations. Both have been free from vertigo and have not had tinnitus or deafness.

*Additions.* Five cases have been added to the group, 3 from True Ménière's Syndrome and 2 from the Atypical Ménière's Syndrome group. Four cases having been removed and five cases having been added, the group now contains 26 cases.

There were 21 original cases retained in this group. Seven of these have died, including 3 since 1949. Death in 6 patients resulted from cerebral hemorrhage and in one from congestive heart failure. The results in the living patients are as follows: 2 Cured; 8 Temporary Relief; and 9 No Relief.

### CORONARY THROMBOSIS

This group originally numbered 13 cases and all have been traced. One case has been reclassified. Case 19 has been free from vertigo since 1949 and has been transferred because the cardiologist has diagnosd the lesion as Coronary Sclerosis.

The ages of the original cases who are alive range from 67 to 76 years. Cases 9, 24 and 164, all retired from business, have not had

recurrence of coronary attacks but only Case 164 is cured of vertigo. A fourth case (36) who continues to work at the age of 76 years, is free from vertigo (cured) but has anginal attacks.

*Additions.* Three cases have been added to this group from Atypical Ménière's Syndrome and 2 cases from Hypertension for reasons already given.

Six of the patients died prior to the 1949 review. Two of the original patients died in 1954. Patient 62 died after repeated attacks of coronary thrombosis. Patient 104 died of cerebral hemorrhage. One patient (167), whose case was transferred to this group from the Atypical Ménière's Syndrome group, died of coronary thrombosis in 1954.

The results in the living patients in this group are as follows: 4 Cured; 3 Temporary Relief, and 1 No Relief.

The group of Coronary Thrombosis which originally contained 13 patients had 1 case transferred and five cases added, so that it now contains 17 cases.

### OTHER CARDIOVASCULAR GROUPS

In the groups designated as Hypotension, Rheumatic Heart Disease and Arteriosclerosis there have been no changes in diagnosis. The present status of these cases in regard to vertigo is indicated in Table I under results. The Coronary Sclerosis group of two cases has been increased by one case transferred from Coronary Thrombosis.

Of the three cases with arteriosclerosis, Case 184 died at the age of 71 from carcinoma of a kidney. Case 136, now aged 77, is cured of vertigo in spite of the fact that in the past five years he has had a right middle cerebral artery thrombosis with paraplegia, evidence of coronary disease, gastric hemorrhage and a prostatectomy.

Case 6, aged 59, diagnosed as rheumatic heart disease, continues to have vertigo and reports the development of a double heart murmur and high blood pressure.

### DISEASES OF THE CENTRAL NERVOUS SYSTEM

There have been few changes in the diagnoses in the group of Organic Neurological Diseases, consisting originally of 27 cases.

Case 146, cured of vertigo, was originally diagnosed Encephalomyelitis but the patient reports that the diagnosis has been changed to Multiple Sclerosis. Case 176, cured of vertigo, was originally diagnosed as Cerebral Atrophy on rather meagre evidence and has been reclassified as Atypical Ménière's Syndrome.

### CEREBROVASCULAR DISEASE

Originally there were two cases in this group. Case 118 died in 1951 at the age of 70 from a heart attack. Two cases have been transferred to this group from True Ménière's Syndrome and have been discussed.

### POST-TRAUMATIC CASES

Originally there were nine patients in this group. One patient has not been traced. Case 151 died in 1950 from malignancy of the spine. It is noteworthy that six of the remaining cases have had no relief from vertigo (Table I).

### BRAIN TUMOUR

There were five patients in this group, all were operated upon and two died prior to 1949. Two of the three remaining patients are cured of vertigo. Patient 100 had an VIII nerve tumor removed from one side in 1947 but in 1950, following a pregnancy, there was recurrence of vertigo and loss of hearing and of vestibular reactions in the opposite ear. Neurological and x-ray examination did not confirm a suspicion of a tumor on that side in 1953. The patient has recently been re-examined. She is unsteady in her gait and is completely deaf. She still has vertigo but has no evidence of intracranial neoplasm.

### NEUROSYPHILIS

There were five original cases and one has not been traced. Two patients, 178 and 197, have had VIII nerve sections performed prior to 1949 and still have occasional vertigo. The other two are cured of the vertigo.

### POSTERIOR INFERIOR CEREBELLAR ARTERY THROMBOSIS

The one patient in this group died in 1950 at the age of 80 years.

TABLE I

| Division of Cases into Groups | Number of Cases in 1949 | Subtractions from Groups Listing Changed Diagnosis | | Additions to the Group | Number of Case not Traced | Number of Cases in 1954 | Results in Reference to Vertigo: Cured | Temp. Relief | Sympt. Relief | No Relief | Dead |
|---|---|---|---|---|---|---|---|---|---|---|---|
| True Ménière's Syndrome | 78 | Toxic Laby. | 8 | | | | | | | | |
| | | Atyp. Mén. Syn. | 8 | 1 | 0 | 55 | 16 | 22 | 4 | 10 | 3 |
| | | Hypertension | 3 | | | | | | | | |
| | | Otosclerosis | 1 | | | | | | | | |
| | | Psychosomatic | 2 | | | | | | | | |
| | | Cerebrovasc. | 2 | | | | | | | | |
| Atypical Ménière's Syndrome | 35 | Toxic Laby. | 4 | | | | | | | | |
| | | Hypertension | 2 | 11 | 1 | 31 | 10 | 6 | 3 | 11 | 1 |
| | | Coronary Thrombosis | 3 | | | | | | | | |
| | | Psychosomatic | 3 | | | | | | | | |
| | | Neurological-Lesion Undiag. | 2 | | | | | | | | |
| Toxic Labyrinthitis | 5 | | | 12 | 1 | 16 | 7 | 6 | 0 | 3 | 0 |
| Coronary Thrombosis | 13 | Coronary Sclerosis | 1 | 5 | 0 | 17 | 4 | 3 | 0 | 1 | 9 |
| Hypertension | 25 | Atyp. Mén. Syn. | 2 | 5 | 0 | 26 | 2 | 8 | 0 | 9 | 7 |
| | | Coronary Thrombosis | 2 | | | | | | | | |
| Hypotension | 2 | | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 2 | 0 |
| Rheumatic Heart Disease | 2 | | 0 | 0 | 0 | 2 | 0 | 1 | 0 | 0 | 1 |
| Arteriosclerosis | 3 | | 0 | 0 | 0 | 3 | 1 | 0 | 0 | 1 | 1 |
| Coronary Sclerosis | 2 | | 0 | 1 | 0 | 3 | 1 | 1 | 0 | 1 | 0 |
| Post-Traumatic | 9 | | 0 | 0 | 1 | 8 | 0 | 1 | 0 | 6 | 1 |
| Brain Tumor | 5 | | 0 | 0 | 0 | 5 | 2 | 0 | 0 | 1 | 2 |
| Neurosyphilis | 5 | | 0 | 0 | 1 | 4 | 2 | 2 | 0 | 0 | 0 |
| Cerebrovascular | 2 | | 0 | 2 | 0 | 4 | 0 | 0 | 0 | 2 | 2 |
| Encephalomyelitis | 1 | Multiple Sclerosis | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Cerebral Atrophy | 2 | Atyp. Mén. Syn. | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| Multiple Sclerosis | 2 | | 0 | 1 | 0 | 3 | 1 | 1 | 0 | 0 | 1 |
| Post Inferior Cerebellar Artery Thrombosis | 1 | | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| Neurological Lesion Undiagnosed | 0 | | 0 | 2 | 0 | 2 | 0 | 1 | 0 | 1 | 0 |
| Otosclerosis | 0 | | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| Psychosomatic | 8 | | 1 | 5 | 0 | 12 | 5 | 0 | 1 | 6 | 0 |
| Totals | 200 | | 46 | 46 | 5 | 195 | 51 | 53 | 8 | 54 | 29 |

### PSYCHOSOMATIC CASES

This group originally contained eight cases. Patient 5 has been transferred to True Ménière's Syndrome and is discussed under that heading. Two cases have been transferred to this group from True Ménière's Syndrome and three cases from Atypical Ménière's Syndrome, and all have been discussed under their appropriate headings. The diagnosis in each case has been confirmed by the internist, a neurologist or a psychiatrist.

There are now 12 cases contained in this group. Seven of these patients have normal hearing, one patient has had an VIII nerve section but has normal hearing in the opposite ear. Seven out of the 12 patients have tinnitus. Five patients are classed as Cured and all are free from tinnitus and 3 have normal hearing. One had Symptomatic Relief and 6 had No Relief.

### TREATMENT

In this review it has been very difficult to form a worthwhile opinion about the value of any treatment because in the replies to the questionnaire the details of treatment in relation to the symptoms leaves much to be desired. It is noteworthy, however, that in the 16 cases of True Ménière's Syndrome classed as cured, 15 were not following any form of treatment. Of the 22 cases in this group classed as Temporary Relief 11 were on a treatment regime which included nicotinic acid and a low salt diet. Of the 10 cases classed as No Relief, 7 were not on any form of treatment.

When describing results in this survey we are merely reporting what has happened to vertigo without any unnecessary relation to treatment. A study of Table I reveals the fact that 70 per cent of the patients with True Ménière's Syndrome have been symptom-free for more than six months whereas only 50 per cent of the patients with Atypical Ménière's Syndrome have been free for the same time. In the group of Toxic Labyrinthitis there was the greatest proportion of cases with relief. Thirteen of the 16 cases had relief for more than six months. In the cardiovascular cases those grouped under Coronary Thrombosis had more relief than those grouped under Hypertension. The mortality has been high in both groups.

The postsurgical cases were appraised in relation to vertigo. There were seven cases in which labyrinthine ablation had been car-

ried out. One died after having been relieved of vertigo for two years. One case was classed as cured.

Of the five remaining cases which were classed as Temporary Relief two had been operated upon in the past five years.

Nine patients had been submitted to complete VIII nerve section prior to 1949. One died a month after operation; a second died four years after section from which there had been no relief. A third case had had no relief and was judged to be psychosomatic. Two cases with neurosyphilis continued to have occasional vertigo. The four remaining cases have been classed as cured.

There have been 29 deaths in the series, 20 from cardiovascular disease. Twelve of the deaths have occurred in the past five years and the average age of these twelve patients at death was 71 years.

In the 1949 survey some consideration was given to a group of 57 patients whose symptoms had been present for more than ten years. It was realized that had that group been assessed in 1944 some cases might have been classed as cured and they could be reassessed in 1949.

Nine of these cases, including six of True Ménière's Syndrome, could have been classed as cured in 1944. One of the nine died of coronary thrombosis in 1945. Of the remaining eight, five remained cured in 1949. Of the eight cases, three have been classed as Cured in 1954, and two of these are cases of True Ménière's Syndrome (Nos. 173 and 193).

## SUMMARY

In the 1949 review there were 200 consecutive cases. We have traced 195 of these in 1954. The original group of True Ménière's Syndrome numbered 78 cases and now this group has been reduced to 55. The original group of Atypical Ménière's Syndrome contained 35 cases which were considered as undiagnosed. Some of these cases developed diagnostic symptoms but not a single case has developed signs of True Ménière's Syndrome (Hydrops).

In classifying results we found that we could not determine the relationship between treatment and symptomatology. As in the 1949 review a cure was taken to mean a period not less than five years free from vertigo irrespective of treatment.

The pertinent literature was very briefly reviewed especially in regard to newer diagnostic criteria.

Twenty-four of 78 original cases of True Ménière's Syndrome have been reclassified. Three developed signs of hypertension, two of cerebrovascular disease and in two cases the signs necessitated transfer to the Psychosomatic group. One case was classed as Otosclerosis because of improvement in hearing after fenestration. In six of the eight cases transferred to Atypical Ménière's Syndrome there was either no tinnitus, no hearing loss or insufficient hearing loss without evidence of progression to justify a diagnosis of Hydrops of the Labyrinth. In one of the remaining cases the hearing loss could have been from causes other than hydrops of the labyrinth and in the eighth case there was loss of consciousness. With regard to the eight cases reclassified under Toxic Labyrinthitis, there had been no hearing loss in three, only a slight hearing loss in two, and a unilateral dip at 8000 cycles in three cases. All have had tinnitus. In all eight cases the symptoms had some relationship to fatigue, emotional strain or infection.

The only addition to this group was the case of a patient who was submitted to labyrinthine ablation for True Ménière's Syndrome and because there was no relief of vertigo, the patient was classed as Psychosomatic. Following a cholecystectomy, nephropexy, and hysterectomy before 1948, the vertigo ceased and has not recurred. In the text a note was made to the effect that this case could be classified as Toxic Labyrinthitis. This case typifies a large number in this series where any one of three or four classifications might be justifiably selected. The point is further emphasized by the fact that in this 1954 review the classification of 46 cases has been changed from that of 1949.

Fourteen of the original 35 cases of Atypical Ménière's Syndrome have been reclassified. None of these patients had any significant hearing loss. Twelve of the 14 cases had no tinnitus or only transient tinnitus. In only two patients was positional nystagmus reported. Two cases were regrouped as Neurological—Lesion Undiagnosed, three as Psychosomatic and three as Coronary Thrombosis. All had normal hearing. All three had single attacks of vertigo, one with and two without tinnitus. Additions to this group were eleven in number; eight from True Ménière's Syndrome, two from Hypertension and one from the Cerebral Atrophy group.

In 1949 the number of cases with severe cardiovascular disease was 47. This number has now been increased to 53. The larger groups are Hypertension 26 and Coronary Thrombosis 17.

There were no significant changes in the Central Nervous System groups.

There was a marked increase in the number of cases in the Toxic Labyrinthitis and Psychosomatic groups. In the reassessment more stress was placed upon the importance of possible toxic and psychogenic factors since these patients did not develop signs of True Ménière's Syndrome (Hydrops) in the intervening five years.

## COMMENTS

It was pointed out in the 1949 review and is again emphasized in this present review that the diagnosis of True Ménière's Syndrome is difficult to establish.

It is interesting that the number of cardiovascular cases in this series now equals that in the True Ménière's Syndrome group and that there are one half as many cases in the Central Nervous System Disease groups. This suggests that when a case of vertigo is under consideration, the chances are equal that the vertigo may be due to True Ménière's Syndrome or to cardiovascular disease, and that there is half as good a chance that the vertigo might be due to a lesion in the central nervous system.

When we have classified a case of vertigo as due to cardiovascular disease we have been aware of the fact that cardiovascular disease and True Ménière's Syndrome may co-exist. We have been especially conscious of that possibility when considering older patients.

It should be pointed out that, whereas not a single one of the original 35 cases of Atypical Ménière's Syndrome which have been traced has developed into a case of True Ménière's Syndrome, 5 cases have developed signs of cardiovascular disease, and 4 of these patients were under 63 years of age.

There were 55 cases of True Ménière's Syndrome representing 27.5 per cent of the 200 cases. Cawthorne and Hewlett on the other hand reported Ménière's Disease in 61 per cent of their series of 1902 cases.

## REFERENCES

1. Cawthorne, Terence, and Hewlett, A. B.: Ménière's Disease, Proc. Roy. Soc. Med. 47:663-670 (Aug.) 1954.

2. Day, K. M.: Surgical Destruction of the Labyrinth for Ménière's Disease, Laryngoscope 62:547-555 (Jan.) 1952.

3. Dix, M. R., and Hallpike, C. S.: The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System, Proc. Roy. Soc. Med. 45: No. 6, 341-354 (June) 1952.

4. Dix, M. R., Hallpike, C. S., and Hood, J. D.: Observations Upon the Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and VIII Nerve, Proc. Roy. Soc. Med. 41:516-526, 1948.

Dix, M. R., Hallpike, C. S., and Hood, J. D.: Nerve Deafness: Its Clinical Criteria, Old and New, Proc. Roy. Soc. Med. 42:527-536, 1949.

5. Eby, L. G., and Williams, H. L.: Recruitment of Loudness in the Differential Diagnosis of End Organ and Nerve Fibre Deafness: Tr. Am. Laryngol., Rhinol. and Otol. Soc. 55:134-148, 1951.

6. Green, R. E., and Douglas, C. C.: Intracranial Division of the Eighth Nerve for Ménière's Disease: The Follow-up Study of Patients Operated Upon by Dr. Walter E. Dandy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:610-621 (Sept.) 1951.

7. Hood, J. D.: Recent Advances in the Study of the Loudness Recruitment and Allied Phenomenon. Acta Oto-Rhino-Laryngol. Belg.: 1, 27-45, 1954.

8. Lindsay, J. R.: The Significance of a Positional Nystagmus in Otoneurological Diagnosis: Laryngoscope, 1945.

9. Lüscher, E.: The Difference Limen of Intensity Variations of Pure Tone and Its Diagnostic Significance: Jour. Laryngol. and Otol. 65:486-510, 1951.

10. McNally, W. J., Stuart, E. A., McKercher, D. D., and Lockhart, H. B.: A Review of Two Hundred Cases of Vertigo, Proc. 4th Internat. Cong. Otolaryng., London, 1949.

11. Nylén, C. O.: Positional Nystagmus. A Review and Future Prospects, Jour. Laryngol. & Otol. 64:295-318 (June) 1950.

12. Reger, Scott N., and Kos, C. M.: Clinical Measurements and Implications of Recruitment, Tr. Am. Otol. Soc. 40:154-167, 1952.

13. Williams, Henry L.: Ménière's Disease, Charles C. Thomas, Springfield, Illinois, 1952.

LVII

# ELASTIC TISSUE OF THE EUSTACHIAN TUBE

STACY R. GUILD, PH.D.

BALTIMORE, MD.

The papers of Reitmann[1] and of Citelli[2] were published half a century ago. Taken together, these two papers afford a good description of the distribution of the elastic type of connective tissue in the walls of the human eustachian tube. The writers of anatomical textbooks and of the anatomical sections of textbooks of otology have, for some reason, overlooked or neglected to utilize the information about this aspect of the tubal structure that has now been available for fifty years. When, even in recent textbooks, any mention at all is made of elastic tissue in the eustachian tube, it is usually merely a statement that the cartilage may contain some elastic fibers. Even in the monographic accounts of the eustachian tube, such as the one eleven years ago by Graves and Edwards,[3] and the one six years ago by Terracol, Corone and Guerrier,[4] the descriptions of the elastic tissue are inadequate. Numerous articles about functional, clinical and pathological aspects of the eustachian tube have appeared in recent decades; in most of them no mention whatever is made of the elastic tissue in the tubal walls.

My purpose in the present paper is two-fold. First, I wish to call attention to the items noted in the above paragraph by presenting some photomicrographs that show the large amount of elastic tissue in portions of the tubal wall. Secondly, I wish to suggest that its elastic tissue may have an important role in the normal functioning of the eustachian tube.

## MATERIAL AND METHOD

The sections used in the present study were stained with Verhoeff's hematoxylin and counterstained with eosin. By this method, elastic connective tissue is given an intense blue-black coloration that

From the Otological Research Laboratory, The Johns Hopkins University School of Medicine.

Fig. 1.—Photomicrograph, at a low magnification, of an adult human eustachian tube section stained for elastic tissue. The section is near the middle of the cartilaginous part of the tube. See text.

makes recognition of all sizes of its fibers easy on direct examination with a microscope even though, in contrast to the identification of elastic tissue with orcein, other tissues are also stained. With photography, however, the differentiation of elastic tissue from other stained elements in the section is not as satisfactory as it is by direct visual examination. Differential filters have not, in my hands at least, excluded entirely the registration on the photographic emulsion of certain other tissue elements stained by the hematoxylin, especially some nuclei and some epithelial cells, red blood cells, calcified cartilage cells and some items in bone. The presence of these other features in

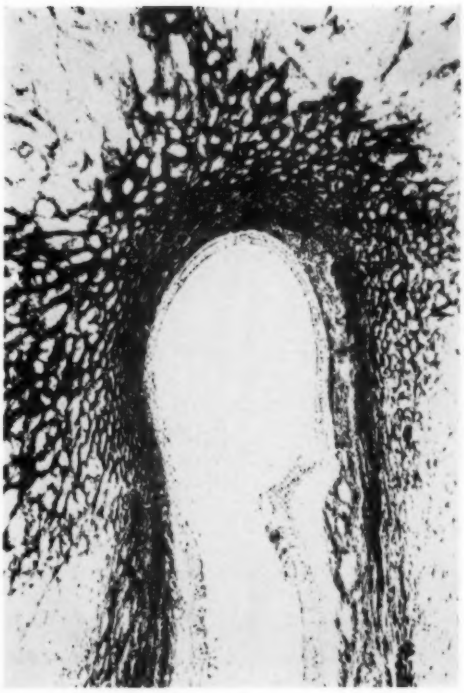


Fig. 2.—A portion of the section shown in Figure 1, at a higher magnification.

the pictures has one advantage, however; it helps orient those who see only the photomicrographs.

The illustrations in this paper are from only three cases, adults aged 39, 57 and 61 years. Tubal sections from 26 human temporal bones have, however, been used in this study. From 10 to 20 sections of each case, at selected intervals along the tube, have been specially stained and examined. Eight of the series used for this purpose are from infants less than one year old; 16 of the series are from adults, aged from 23 to 65 years.

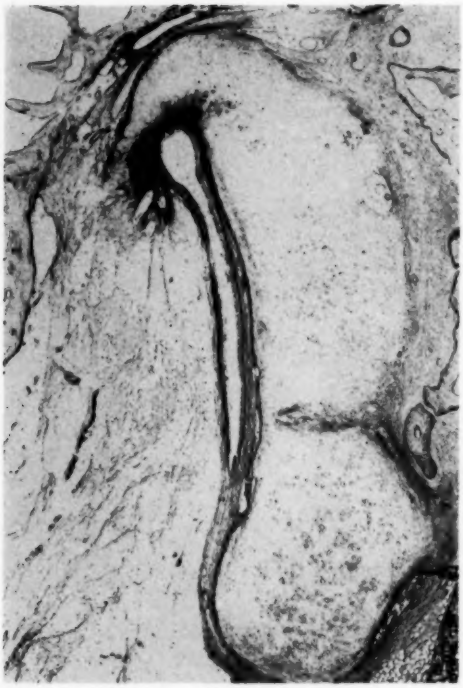


Fig. 3.—Photomicrograph, at a low magnification, of an adult human eustachian tube section stained for elastic tissue. This section is from a different case than the one shown in Figure 1 and is from a level closer to the tubal isthmus.

## OBSERVATIONS

In the cartilage of the eustachian tube the elastic type of fibrous tissue is especially abundant in the inferior part of the region of continuity of the medial and lateral plates. Here the quantity of elastic fibers in the ground substance, or matrix, of the cartilage is so great that in sections cut transversely to the long axis of the tube the densely stained area has the appearance of a cap or of a corona over the superior part of the tubal lumen; the dense area extends somewhat farther into the lateral than into the medial plate (Figs. 1,

2 and 3). The perichondrium of the tubal cartilage has a considerable content of elastic tissue in all regions (Figs. 1 and 3), but in the part adjacent to the aforementioned "cap" of the elastic cartilage the perichondrium is especially rich in this type of connective tissue. In this region no line of demarcation can be recognized under the microscope between perichondrium and cartilage or between perichondrium and submucosa, which layer also has a high proportion of elastic fibers in this region (Fig. 2).

The elastic tissue in the mucosal and submucosal layers of typical regions of the medial and lateral walls of the cartilaginous part of the eustachian tube is portrayed in Figure 3, and at a higher magnification in Figure 4. The mucosal layers of elastic tissue, as may be seen in these illustrations, is thicker and more dense in the lateral, or membranous, wall of the tube than in the medial, or cartilaginous, wall, while the reverse is the case with the submucosal elastic tissue. Figure 4 also shows that the elastic tissue content and density of the mucosal layer of the medial wall is about the same as that of the perichondrium of the adjacent region of the cartilage. Over the free, or convex, surface of the cartilage the elastic tissue of the perichondrium is more loosely arranged than it is in the region adjacent to the lumen of the tube (Fig. 3).

In the osseous part of the eustachian tube it is usually not possible to make a clear distinction between the mucosal and submucosal layers of elastic tissue (Fig. 5); typically the two are blended together into a layer so rich in elastic tissue that even at the higher magnifications photomicrographs record it only as a solid band (Fig. 6). It is of interest to note that this dense layer of elastic tissue does not extend, even as a thin loose layer, into pneumatized cells that open from the tubal lumen; instead, the layer of elastic tissue stops abruptly at the orifice of the cell (Figs. 5 and 6).

## POSSIBLE FUNCTION

From sections cut approximately parallel to the long axis of the cartilaginous tube through the dense submucosal layer above its lumen (sections not illustrated here), also from the transversely cut sections, I have the impression that a large proportion of the coarser elastic fibers of this region run almost longitudinally. Because the fibers cannot be followed in a section for any considerable distance, I cannot be certain whether the further impression that such fibers, or the

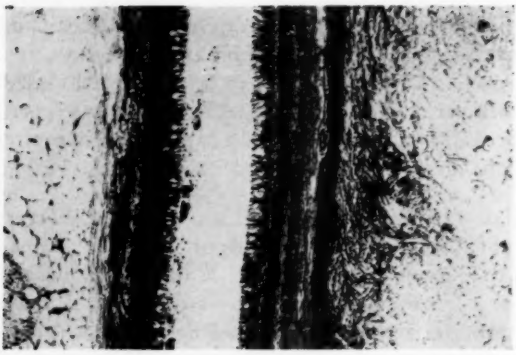


Fig. 4.—A portion of the section shown in Figure 3, photographed at a higher magnification. The lateral wall of the tube is toward the left. See text.

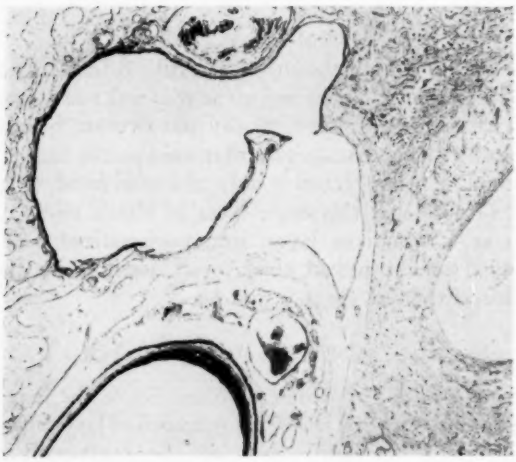


Fig. 5.—Photomicrograph, at a low magnification, of a section through the tympanic expansion of the osseous part of the eustachian tube, stained for elastic tissue, at the mouth of an infratubal cell. Human adult. The picture includes, for orientation, a portion of the basal turn of the cochlea and a bit of the wall of the internal carotid artery, ascending part, in the carotid canal.

Fig. 6.—Shows, at a higher magnification than in Figure 5, the abrupt cessation of the tubal elastic tissue at the mouth of the infratubal cell.

networks they form, also have a spiral course laterally and downward, is correct or not. If the impression is correct, and if the heavy bands of elastic tissue that can be seen in Figures 1 and 3 lateral to the upper part of the tubal lumen are thus oriented, it seems at least a plausible speculation that this elastic tissue, together with that of the "cap" of the cartilage itself, has a physiologic role in counterbalancing the action of the tensor veli palatini muscle. As is well known, this muscle is attached in part to the lateral plate of the tubal cartilage and in part to the membranous segment of the tubal wall. The latter attachment is by means of numerous tiny tendinous strands

that traverse, in an obliquely upward direction, the soft adipose tissue between the main part of the muscle and the tube. Contraction of the tensor veli palatini muscle thus pulls the membranous lateral wall of the tube away from its relatively stationary medial wall and opens the tubal lumen. Closure of the tubal lumen, and maintenance of its usual condition as only a potential lumen, has usually been regarded as a passive affair, caused merely by the pressure of adjacent structures. I suggest that the structural facts presented today, and which have been known in essentially the same form for half a century, constitute evidence that indicates the closure of the tubal lumen, after it has been opened by contraction of the tensor veli palatini muscle, also the maintenance of its normally closed condition, may be an active instead of a passive phenomenon. In other words, my suggestion is that there is evidence to indicate the rapid closure of the tube which normally follows any opening of its lumen during swallowing or yawning, may be a rebound action of the elastic tissue of the "cap" of the cartilage and of the other abundant elastic tissue of the region, which has been stretched by the muscular contraction that opened the tube. Furthermore, it seems reasonable to believe that the heavy layer of elastic tissue of the mucosa of the lateral wall may play an important part in the maintenance of the tonus of the closed tubal lumen.

One further morphologic item lends support to the functional suggestion offered; namely, in young infants the amount of the elastic type of connective tissue in the tubal walls is much less than is present in older children and in adults. Perhaps a relation exists between the amount of elastic tissue of the tubal walls, at different ages, and the incidence of otitic infections via the lumen of the eustachian tube.

SUMMARY

The distribution of the elastic type of connective tissue in the walls of the human eustachian tube is in part described and illustrated by photomicrographs. The structural facts indicate that the elastic tissue may have an important role in the normal functioning of the tube, especially in the prompt closing of the lumen and in the maintenance of its closure.

JOHNS HOPKINS UNIVERSITY.

## REFERENCES

1. Reitmann, K.: Ueber den Bau des Tubenknorpels beim Menschen. Monatschr. f. Ohrenheilk. 37:45-50 (Feb.) 1903.

2. Citelli, S.: Sulla struttura della tromba d'Eustacio nell uoma. Arch. ital. di Otologia, etc. 16:404-418 and 441-452, 1905.

3. Graves, G. O., and Edwards, L. F.: The Eustachian Tube; A Review of Its Descriptive, Microscopic, Topographic and Clinical Anatomy. Arch. Otolaryngol. 39:359-397 (May) 1944.

4. Terracol, J., Corone, A., and Guerrier, Y.: La Trompe d'Eustache. Masson & Cie., Paris, 1949. 218 pages.

LVIII

# CHEMODECTOMAS OF THE GLOMUS JUGULARE (NONCHROMAFFIN PARAGANGLIOMAS) WITH ESPECIAL REFERENCE TO THEIR RESPONSE TO ROENTGEN THERAPY

HENRY L. WILLIAMS, M.D.

DONALD S. CHILDS, JR., M.D.

EDITH M. PARKHILL, M.D.

AND

DAVID G. PUGH, M.D.

ROCHESTER, MINNESOTA

In 1941 Guild[1] described a structure which he called the "glomus jugularis." He stated that this structure was histologically like the carotid body, being composed essentially of blood vessels of capillary or of precapillary caliber with numerous epithelioid cells between the vessels.

In a paper in 1953[2] he described this structure more fully, basing his description on histologic sections of 88 temporal bones (both ears of 44 persons). He found that jugular bodies occur with almost equal frequency along the courses of the nerve of Jacobson (tympanic branch of the glossopharyngeal nerve) and of the nerve of Arnold (auricular branch of the vagus). He stated that somewhat more than half of the glomus formations were found in the adventitia of the dome of the jugular bulb and another fifth in the osseous canal through which the nerve of Jacobson enters the middle ear from the jugular fossa. About one fourth are in the mucosa of the promontory and some are along the course of the nerve of Arnold beyond the jugular fossa.

Guild[2] stated that the differences in clinical symptoms reported by patients with glomus jugulare tumors and also in the apparent

From the Mayo Clinic and the Mayo Foundation, Rochester, Minn.

sites of origin found at operations for such tumors can all be satisfactorily explained by the normal differences in the locations of jugular bodies and by any abnormal enlargement of these bodies.

In 1945 Rosenwasser,[3] while studying sections of a tumor of the middle ear on which he had operated, recalled the report of Guild and suggested that the tumor had arisen from a "glomus jugularis" rather than from the carotid body, which it resembled histologically.

In 1949 Lattes and Waltner[4] reported on the pathologic findings in eight cases of carotid-bodylike tumor. They pointed out that the correct Latin diction for Guild's term should be "glomus jugulare." In preference to the unsatisfactory term of "carotid-bodylike tumors" they suggested that these tumors be designated "nonchromaffin paragangliomas."

Mulligan[5] proposed the term "chemodectoma" as a more descriptive designation of neoplasms arising from paraganglionic tissue. He defined chemodectoma as a neoplasm consisting of chemoreceptor cells which are associated with the distribution of parasympathetic nerves and which originate in the adventitia of blood vessels in structures intimately connected with afferent nerve fibers or along the branches or in the ganglia of the glossopharyngeal nerve. Because of its shortness and convenience we had adopted this term to describe a tumor originating from one of the glomus jugulare structures described by Guild. The recent report by Huppler, McBean and Parkhill[6] suggests that these tumors may have physiologic function.

In 1951 Dockerty, Love and Patton[7] reviewed the literature and reported a case of nonchromaffin paraganglioma of the middle ear in which the clinical aspects were those of a brain tumor.

In 1954 Figi and Weisman[8] again reviewed the literature and reported the results obtained in 22 patients operated on for hemangioma or hemangio-endothelioma. The histologic sections in these cases were restudied and each lesion was found to be chemodectoma (nonchromaffin paraganglioma) of the glomus jugulare.

## MALIGNANCY OF CHEMODECTOMAS

In none of the material studied by Figi and Weisman was metastasis discovered. They were of the opinion that the two instances of metastasis reported in the literature were probably instances of simultaneous occurrence of chemodectoma and carotid-body tumors, several instances of which had been reported.

Lattes and Waltner,[4] however, reported a case of metastasis in the liver. They stated, "A section from the liver metastasis shows the neoplastic tissue to be poorly preserved due to necrosis and post-mortem changes. However, it is essentially similar to the tumor observed in the cranial cavity."

Since metastasis has not been reported in any of the later cases in this country and abroad, this identification may be viewed with reasonable doubt. The consensus of other observers is that these tumors are malignant only because of their possible encroachment on intracranial structures and that they do not metastasize. They should, therefore, apparently be classified as benign neoplasms.

## SYMPTOMATOLOGY

Lattes and Waltner[4] stated that the symptoms of chemodectoma of the glomus jugulare are nonspecific, that is, they do not differ from those produced by any slowly growing tumor in the middle ear and its environs. This was also the opinion of Rosenwasser,[9] and Brown[10] was not able to discover any sign or symptom characteristic of this particular lesion. The only suggestive finding that these authors mentioned is the tendency to excessive bleeding on slight trauma.

## CLASSIFICATION OF CHEMODECTOMAS

Riemenschneider and associates[11] classified glomus jugulare tumors as either benign or malignant. This however is not a histologic classification. They stated that a benign tumor may or may not represent an earlier stage of a malignant tumor. Malignant change they felt was characterized by massive destruction of the petrous pyramid, involvement of cranial nerves by pressure on the brain stem or a mass in the nasopharynx.

Capps[12] stated that these tumors appear to fall naturally into two groups. The first group is comprised of those tumors arising in the middle ear. These give rise to little in the way of symptoms except deafness and tinnitus and present through the drumhead into the external auditory meatus. In the second group are those tumors arising in the neighborhood of the jugular bulb and invading the middle ear through its floor. These often tend to invade the labyrinth and to spread on the under surface of the petrous bone giving rise to cranial nerve palsies. These tumors are often so extensive and

inaccessible that surgical approach is a matter of great difficulty and fraught with considerable danger to the patient.

## TREATMENT

In the earlier papers surgical treatment of chemodectomas of the glomus jugulare was stressed. Weille and Lane[13] did not mention any other type of treatment. Rosenwasser stated that it is becoming increasingly apparent from the available opinion expressed that radiation even as administered today unaccompanied by surgery offers little of encouragement. Barton and Thee[14] stated that radiation therapy has produced no demonstrable effect. Mattick and Burke[15] stated that there seems to be no reason for radiation of glomus tumors as presumably, like carotid-body tumors, they are radioresistant. They qualified this by mentioning that Graf[16] had reported favorable response to deep x-ray therapy in eight cases. Bradley and Maxwell[17] favored surgical treatment of chemodectomas of the glomus jugulare although two of their patients were treated with roentgen therapy alone. They stated that although clinical and roentgenographic evidence indicates little response to this treatment, both of these patients were living 15 years after treatment. Whether or not the natural slow growth of the tumor had been retarded they did not know.

Riemenschneider and co-workers[11] stated that although there is little evidence that radiation therapy accomplishes much, the few reports of fair results warrant its use in cases of obvious inoperability.

Capps[12] stated that in extensive cases radiotherapy may be the treatment of election. He found it effective also in cases of local recurrence of the localized type of tumor when the previous treatment had been a radical mastoid operation. Strong[18] stated that in the event that removal has been incomplete, recurrence is inevitable.

Brown[10] stated that the therapy of glomus jugulare tumors should include surgical removal or x-ray therapy or both. In his last case he reported that the treatment consisted of x-ray therapy alone.

From this review it seems apparent that an early enthusiasm for surgical treatment is on the wane and that more recently it is felt that roentgen therapy must be added to obtain a good result or that radiotherapy alone may be sufficient to control the lesion.

## MATERIAL STUDIED

Thirteen patients given the diagnosis of chemodectoma of the glomus jugulare at the Mayo Clinic are included in this study. Two

of these patients were included in the review of Figi and Weisman, but because they seem to illustrate the effect of radiation therapy they are again reviewed.

In two cases biopsy was not obtained at the Mayo Clinic. The diagnosis of chemodectoma was made from the symptoms and signs, the roentgenographic appearance of the lesion and the clinical course of the disorder.

## REPORT OF CASES

CASE 1.—This patient was first seen at the clinic in March, 1952. Three years previously, because of ringing in the right ear, a small growth apparently arising from the floor of the external auditory canal had been found and excised. The pathologic report was "sclerosing hemangioma." Three months later the growth began to recur. Hearing in the right ear rapidly disappeared in 1950. A short time before this the patient had a sensation of swaying when she walked. This disappeared after a time. Twitching in the right side of the face had begun in August, 1951, and rapidly developed into weakness. For a few months before coming to the clinic she had a right frontal headache on arising but this disappeared when she took one or two aspirin tablets.

On examination of the right ear at the clinic a rounded, soft reddish mass, the size of a pea, covered with unbroken epithelium and easily indented, was seen in the external auditory meatus. This obscured the canal and ear drum, although it appeared to be attached to the floor of the canal. There was no discharge from the ear. The right side of the face was partially paralyzed with flattening of the wrinkles on the right. The tongue on protrusion deviated toward the right.

A pure-tone audiogram showed normal hearing in the left ear by air conduction. In the right ear, tones of 512, 4096 and 8192 frequencies were heard at 95 decibels but other tones were not heard.

Our modification of the Kobrak test showed a normal reaction in the left equilibrial labyrinth and complete lack of response in the right.

Roentgenograms of the mastoid processes and head showed destruction of the tip and inferior aspect of the petrous portion of the right temporal bone. Extensive destruction of the adjacent floor of the posterior fossa and some destruction of the floor of the middle fossa anterior to the right mastoid process were evident. The mastoid cells were indistinct and some of the cell walls appeared to have been destroyed.

Vision, pupils, ocular reflexes, fundi and visual fields were essentially normal on both sides.

Weakness of the right VII and XII cranial nerves and weakness with fibrillation on the right side of the tongue were noted, sensation of right VIII cranial nerve was −4, right corneal anesthesia was −1, and there were occasional jerking nystagmus in the right eye on extreme left lateral gaze and occasional unsteadiness on quick turning. There was no evidence of increased intracranial pressure.

A diagnosis of inoperable chemodectoma (nonchromaffin paraganglioma) of the right glomus jugulare was made and intensive roentgen therapy was advised. A

tumor does of 3,700 roentgens was delivered through three fields using 250 kv with HVL of 1.3 mm copper over a period of 12 days and the patient was dismissed.

In March, 1955, her physician reported that no change was visible in the extensive bone defect in the skull on the right side when compared to roentgenograms taken in February, 1952.

CASE 2.—A woman, aged 33 years, presented herself to us in 1947. She had been perfectly well until she had the "flu" in the spring of 1932. A pounding noise synchronous with the heartbeat developed somewhere in her head about a month later. At first it was noticeable only at night but in three or four months became constant day and night. After a few inflations of the eustachian tubes the tinnitus localized in the left ear. At about this time hearing in her left ear began to decrease. In 1934 tonsillectomy and adenoidectomy were done and tubal inflations were continued because of loss of hearing. In 1935 left myringotomy was done. There was a discharge of blood lasting for only a few minutes. A few months later sphenoidostomy was done, but the loss of hearing became complete about this time. In 1935 the patient was told that a crust had formed over the left ear drum and she could feel something in the left ear canal with her finger. In 1936 a persistent purulent discharge began from the left ear canal and granulation tissue appeared in the external auditory canal. In the spring of 1938 the granulations in the left ear canal were cauterized. The discharge from the ear continued and slight dizziness and nausea developed. A severe attack of vertigo and nausea confined her to bed for two weeks in August. In December, 1938, a left mastoidectomy did not improve her symptoms. She continued to have slight dizziness and nausea. In January, 1941, the left eyelid did not close completely and in February jerking movements of the left eyelid were noted. Weakness gradually spread and by September, 1941, paralysis of the left side of the face had become complete. A third operation was done on the left mastoid, at which time the left facial nerve was "uncapped." Movement in the left side of the face returned partially but was lost again by December, 1941. In January, 1942, severe left-sided headaches developed and another mastoidectomy was attempted. Symptoms continued.

In November, 1946, a large granulomatous mass was removed from the left external auditory canal and another mastoidectomy was attempted. Profuse bleeding from soft tissues and bone caused shock on the operating table even though the patient was receiving blood intravenously at the time.

After repeated transfusions the left internal jugular vein was ligated and an umbilical tape was placed around the left common carotid artery. Mastoidectomy was again attempted with an infusion of blood into both arms and one leg. Again the operation had to be discontinued because of bleeding. Occlusion of the left carotid artery by pulling on the umbilical tape had no effect on the bleeding, nor did exposing the lateral sinus back toward the torcular and packing it off. Some material was removed from the antral and attic areas.

Symptoms of nausea, vomiting and headache soon returned and in May, 1947, she was referred to the Mayo Clinic.

Examination disclosed a bulging mass covered with scar tissue which closed off the left external auditory canal lateral to the isthmus. A bruit over the left postauricular area, synchronous with the pulse beat, could be stopped by compression of the left carotid artery but not by occlusion of the right carotid. The tongue on protrusion deviated toward the left, and the soft palate was weak on the left side. There was loss of taste on the left side of the tongue and the left vocal cord was in midposition. The ocular fundi were negative. Neurological examination showed

involvement of the left VII to XII cranial nerves. Roentgenograms of the mastoids showed a mixed pneumatic type of cell on the right. The left mastoid showed evidence of surgical interference with extensive destruction of the remaining cells. Cells of the left petrous pyramid appeared completely destroyed.

Tissue removed at operation elsewhere was reported to show a hemangio-endothelioma.

The condition was considered inoperable. Radium treatment was given on four consecutive days, with a total dose of 1,100 roentgens to the lesion on the left.

Four months later movement was present at the angle of the mouth and nasal ala on the left but no other change was visible to the eye or in the roentgenograms.

Further radium therapy was given in September, 1947, and in February and September, 1948; a total of 2,200 roentgens then had been delivered to the tumor-bearing region.

Chronic nervous exhaustion developed, probably owing to the long course of her illness and the death of her 8-year-old son from leukemia. Burning pain developed in the left side of her face and left arm. Comparison of the roentgenograms taken in August, 1948, with those taken previously did not show much increased destruction in the petrous pyramid on the left. Stereoscopic views of the base of the skull, which had not been taken previously, now showed rather extensive destruction of the floor of the posterior and middle fossae adjacent to the petrous pyramid.

At the patient's last visit in August, 1949, she was feeling well and had gained weight. She was free of headache, dizziness and nausea. The left external auditory canal was partially occluded by a scar. Through a perforation in the scar a small portion of the ear drum could be seen. The cranial nerve palsies had not improved further. Since growth of the tumor had been arrested, no further treatment was indicated. A recent letter from the patient stated that her condition was subjectively the same as when seen here in 1949.

CASE 3.—In 1950 a thumping developed in the right ear of this patient, a woman aged 50 years, during an attack of "flu," and gradually grew louder. In 1952 a biopsy elsewhere proved a mass in the right ear to be a nonchromaffin paraganglioma of the glomus jugulare. Continuous rusty, yellow or clear fluid drainage began from the right external auditory canal.

On our examination at the clinic in 1953 the right external auditory canal was found to be blocked by a reddish, friable, raspberrylike mass. A roentgenogram of the mastoids including Stenver's projection revealed partial sclerosis of the right mastoid, the left mastoid being negative.

At radical mastoidectomy through a postauricular incision the mastoid cells were found filled with a serosanguineous fluid. The mastoid was completely exenterated but no bleeding was encountered until the region of the jugular bulb was approached. The retrofacial group of cells was very large and joined with a group from the hypotympanum. The posterior wall of the canal, the bridge and the lateral wall of the epitympanum were then removed, exposing the middle ear, which was filled with a friable, easily bleeding mass. This was removed as completely as possible and the region was packed to control bleeding. The patient was given 500 cc of whole blood during the operation. Radium tubes were packed into the wound and after operation external radium therapy was used at a distance of 5 cm. A total dose of 1,800 roentgens was delivered to the involved area.

The patient was seen in 1954. The cavity was in good condition, but there was a small area of granulation tissue over the promontory. A roentgenogram of the mastoids taken at that time showed a postoperative defect in the right mastoid.

CASE 4.—A woman, aged 50 years, was referred to us in September, 1952, because of a mass protruding from the left external auditory canal. In 1948 she had no complaints referable to the ears. In 1950 she noticed a thin discharge from the left ear and swelling of the left external auditory canal.

About a week before her admission to the clinic pain deep in the left ear developed. In the daytime this pain was shooting and stabbing, at night an aching pain. On the second and third days before admission she had "shaking chills" in the evening.

Examination disclosed that the left external auditory canal was filled with a friable, easily bleeding mass, apparently originating from below. This resembled a large aural polyp. An audiogram showed a bilaterally symmetrical perceptive deafness. A roentgenogram of the mastoids showed sclerosis of both mastoids with a possible cholesteatoma on the left.

Biopsy of the mass protruding from the left external auditory canal revealed chemodectoma.

At operation the middle ear and mastoid antrum were found filled with a granulationlike tissue which was removed as completely as possible. A fresh frozen section of this material also revealed a chemodectoma (nonchromaffin paraganglioma) of the glomus jugulare. Little more than the average amount of bleeding was encountered. After the radical mastoidectomy was completed in the usual manner radium tubes in the wound at a distance of 5 cm delivered a dose of 4,000 roentgens to the tumor-bearing tissues.

The patient was examined three and nine months after dismissal. At present, 15 months following operation, her home physician reports her free of signs or symptoms of any disorder in connection with her left ear.

CASE 5.—Following an earache in 1913 the right ear had begun to discharge and this had continued. On admission to the clinic in 1915 there was a moderate purulent discharge in the right external auditory canal, which was choked with granulation tissue and epithelial debris. The patient, a woman 23 years old, heard a loud spoken voice at the right ear while a soft whisper was heard at three feet on the left.

The granulations were removed and their base cauterized with 50 per cent solution of silver nitrate. A month later right radical mastoidectomy was done. Profuse bleeding occurred from the middle ear.

Drainage from the right ear had started again about three months before admission in December, 1920. About a month before admission, during a cold in the head, the right side of the face felt a little stiff and the right eye could not be closed completely. Neurologic examination disclosed weakness on protrusion of the tongue, as well as facial palsy on the right. The disease in the right mastoid seemed to involve the VII and XII cranial nerves.

A purplish-red fluctuating mass covered the upper portion of the right middle ear and external auditory canal. A vascular tumor was suspected because of the profuse bleeding which had followed incision.

Treatment cleared up the discharge from the ear for nine months. Then the right postauricular region began to swell and in three weeks the ear was bulged

forward and for a week the patient had severe pain in the right mastoid region, especially on lying down.

On examination a red fluctuating swelling bulging the region of the previous mastoidectomy scar was found. A large bluish mass filled the upper portion of the cavity and the external auditory canal.

The right mastoid was explored. Separation of the soft parts was accomplished without severe bleeding, but a profusely bleeding mass filled the antrum and region of the tegmen. Biopsy showed inflammatory granulation tissue.

The patient had no further trouble until June, 1924. Then shooting pains developed over the right side of the head, referred to the right eye. Facial palsy was still present and the mass in the right external auditory canal had changed little. The right nasal chamber was blocked by a polypoid mass and a rounded mass in the nasopharynx surrounded the orifice of the right eustachian tube.

Seven courses of external radium therapy were given in 1924 and 1925. Biopsy in 1931 revealed fibrosarcoma, Grade 4. Between 1931 and 1933 external x-ray therapy was administered to the nasopharynx with a total dose to the nasopharynx of about 3,500 roentgens being delivered using 135 kv. Both her local and general condition improved.

The patient returned on numerous occasions and in 1940 the deformity of the right face was corrected.

In 1944 an area of necrotic bone became visible in the middle ear cavity and roentgenograms of the mastoids showed marked destruction of the temporal bone in the region of the right mastoid process with involvement of the entire petrous pyramid. Further external radium therapy was administered to the right mastoid.

Through a postauricular incision a large quantity of necrotic bone was removed. Biopsy revealed inflammatory and degenerating tissue.

The patient then was symptom free as far as the ear was concerned. She died in 1946 of unknown cause.

**Owing to the unprecedented good result, the tissue was restudied in 1952 and an amended report of chemodectoma with marked anaplasia of cells imitating a fibrosarcoma was made.**

CASE 6.—A 49-year-old woman consulted us on July 8, 1954. For 20 years she had had occasional attacks of acute otitis media, always on the right side. About a year before admission an attack of acute otitis media was followed by a persistent discharge of foul yellow purulent secretion.

About nine months before admission biopsy revealed a polypoid hemangiomatous benign fibroma in the right external auditory canal. The mass was removed on several occasions; its base was cauterized with both silver nitrate and chromic acid and finally with electrocautery, but recurrences continued. The patient had no pain but deafness increased with a pounding tinnitus synchronous with the pulse.

The right tympanic membrane was bulging and beefy red. The audiogram showed a moderate degree of conduction deafness on the right side. Roentgenograms showed extensive sclerosis of the right mastoid and a negative left mastoid.

Sections of tissue removed elsewhere were forwarded to one of us (Parkhill) and a diagnosis of chemodectoma was made.

The mastoid was explored through a postauricular incision so that the tumor in the middle ear could be approached from behind and the larger part of the mastoidectomy accomplished before bleeding from the chemodectoma would be encountered.

After operation 1,600 roentgens was given to the right mastoid and middle ear using 250 kv with an HVL of 1.3 mm copper.

On examination in December, 1954, the mastoid cavity was completely epithelized and dry. A roentgenogram of the mastoid revealed postoperative changes only.

CASE 7.—In 1950 the patient had sudden pain in the right ear which cleared up without rupture of the drum or drainage on treatment with penicillin. About two weeks later she noted the gradual onset of deafness in the left ear without pain. She lost 90 per cent of her hearing for conversation in the first month or two but believed that since then there had been no increase in the deafness.

On examination at the clinic in June, 1951, a reddish-pink region was seen in the lower portion of the left ear drum reaching up to the umbo. A red mass in the middle ear seemed to be pushing through the drum membrane. The rounded border of the mass in the middle ear could be made out on compression. Inflation of the left eustachian tube caused no change in the appearance. The audiogram was as follows:

| | 125 | 256 | 512 | 1024 | 2048 | 4096 | 8192 |
|---|---|---|---|---|---|---|---|
| AC, right | 15 | 10 | 5 | 5 | 10 | 25 | 20 |
| left | 45 | 55 | 50 | 55 | 65 | 75 | |
| BC, right | | 30 | 30 | 20 | 25 | 30 | |

There was no response to bone conduction testing in the left ear with masking at 80 decibels which was required for proper lateralization.

Roentgenograms of the mastoids showed mixed cell pneumatic mastoids but no pathologic change.

The tentative diagnosis of chemodectoma (nonchromaffin paraganglioma) of the glomus jugulare on the left was confirmed by biopsy. A 50 mg tube of radium enclosed in a Penrose drain was placed against the drum and left in place for two hours and fifteen minutes.

Three months later the left tympanic membrane appeared thickened with a small bead of granulation tissue at the umbo. The tongue deviated toward the left on protrusion and the left side was atrophied with some fasciculation. The left side of the pharynx was weak and also had impaired sensation. The left vocal cord was fixed in the midline.

Vision was normal in both eyes as were the pupils, reflexes and visual fields. There was an alternating divergent strabismus of five degrees. The ocular fundi showed normal disks and maculae and retinal sclerosis Grade 1 (on the basis of 1 to 4).

Roentgenograms of the mastoids showed a change in the left mastoid in the last three months. There was diffuse haziness in the mastoid cells on the left but no destructive process. A roentgenogram of the head was negative.

Neurologic examination revealed involvement of the IX to XII cranial nerves on the left and a diagnosis of a jugular foramen syndrome was made.

A tumor dose of 1,440 roentgens was delivered, using 200 kv with an HVL of 0.98 mm copper at a distance of 50 cm.

The patient returned three and also six months later. The condition of the tongue and larynx was unchanged but the movement of the palate and sensation in the pharynx were normal. The patient subjectively was much improved. No progression of the lesion was noted since the roentgen therapy six months before.

CASE 8.—This patient, a woman aged 45 years, was first seen in April, 1948. Twelve years previously acute otitis media on the left had developed during an acute upper respiratory tract infection. Thick yellow pus drained for a few weeks and then cleared up under local treatment. She had recurrences of the discharge with each subsequent head cold. In August, 1947, she had a marked exacerbation with swelling over the mastoid region and tenderness on pressure. The acute symptoms cleared up but the left ear continued to discharge. In February, 1948, removal of a polyp pathologically diagnosed hemangioma from the left ear was accompanied by profuse bleeding. Deafness had increased on the left and had become complete three years before admission to the clinic. There had been no evidence of involvement of other cranial nerves.

On examination a pale, smooth, rounded tumor mass was seen filling the right external auditory meatus almost to the conchal opening. Complete deafness was found on the left. Roentgenograms of the mastoids showed periantral sclerosis and cloudiness of the mastoid cells on the left, the right mastoid being normal.

The growth removed from the left external auditory canal under pentothal anesthesia was considered to be a hemangio-endothelioma, Grade 1. In 1952, however, it was rediagnosed a chemodectoma (nonchromaffin paraganglioma) of the glomus jugulare.

X-ray therapy was given in April, July and November, 1948. A tumor dose of 1,230 roentgens was given over this period using 135 kv with HVL of 0.3 mm copper at a distance of 40 cm. Re-examination six and nine months later showed that the tumor was reduced to a small fibrous, smoothly epithelized mass in the middle ear without any tendency to bleed on manipulation.

Roentgenogram of the mastoids taken nine months after treatment revealed no appreciable change since the previous examination.

In October, 1951, the canal was filled with epithelial debris. After removal of this material a tender, soft mass was found on the superior wall of the external auditory canal. A glomus tumor was suggested. Roentgenograms of the mastoids showed no change since previous examinations.

The patient received a tumor dose of 1,000 roentgens, using the radium at a distance of 5 cm from the skin at that time, and in March, 1952, 500 roentgens was given by the same means.

By April, 1953, the mass on the superior wall of the left external auditory canal was reduced to a small, scarred mass. Roentgenograms of the mastoids showed no change. A radon seed, 1.34 mc, was placed in the center of the fibrous mass, and produced considerable bleeding. The patient reported in 1954 that she had no symptoms in reference to the left ear.

CASE 9.—A man, aged 47 years, consulted the clinic in June, 1953. During an infection of the upper respiratory tract in 1950 the right ear suddenly became partially deaf and there was a continuous ringing and hissing sound in the ear. He had no pain in or discharge from the ear and no vertigo or dizziness. Inflations of the right eustachian tube relieved the hissing sound but did not lessen the ringing or deafness.

Tonsillectomy was performed in 1951 because of generalized arthritic pains. Relief was obtained.

Eight weeks before his admission a sensation of pressure developed in the right ear on coughing, accompanied by a sensation as if he were about to fall toward the left. Occasional sharp pain was felt in the region of the right mastoid process.

On examination the right ear drum appeared red and irregular and none of the usual landmarks could be made out. The ear drum seemed to be pushed out by something behind it. The tongue on protrusion deviated toward the right, there was weakness of the right side of the palate and the right vocal cord was in the cadaveric position. With high-level masking of the left ear there was apparently no sound reception in the right ear and the equilibrial labyrinth on the right appeared to be without function.

Roentgenograms showed diffuse sclerosis of the right mastoid with some cellular destruction in the region of the antrum. The ocular fundi were negative.

A diagnosis was made of probable chemodectoma of the middle ear and mastoid on the right with jugular foramen syndrome.

Through a postauricular incision the right mastoid was opened. The cells contained an amber-colored fluid and the bone bled freely. The mastoid cells were completely exenterated and the posterior bony wall of the external auditory canal and the bridge were removed. An easily bleeding tumor mass filled the middle ear and extended through the middle ear into the antrum. This tumor was removed and proved to be a chemodectoma (nonchromaffin paraganglioma) of the glomus jugulare on pathologic examination.

After operation a tumor dose of 1,400 roentgens was given through a single 8-cm circular field directed at the middle ear; 200 kv with an HVL of 0.98 mm copper was used at a distance of 50 cm.

Facial weakness developed on the right side but this had almost completely disappeared three days later.

Three months later there was no gross evidence of tumor and the mastoid cavity was healing satisfactorily. A year later there was no evidence of local recurrence of the tumor. The jugular foramen syndrome remain unchanged.

CASE 10.—In 1943 in the course of a general examination and without any history of symptoms referable to the ears, a reddish mass was seen lying behind the left tympanic membrane. A slight hearing loss was found on the left.

The patient returned seven months later. She had noticed some weakness at the left angle of the mouth shortly after the first visit. This had gradually increased until one month before returning it had completely involved the entire left side of the face.

The appearance of the left ear had not changed. The audiogram was as follows:

| | 125 | 256 | 512 | 1024 | 2048 | 2896 | 4096 | 5192 | 8192 |
|---|---|---|---|---|---|---|---|---|---|
| AC, right | 5 | 5 | 5 | 0 | 0 | 5 | 5 | 0 | 0 |
| left | 25 | 10 | 40 | 40 | 40 | 50 | 50 | 30 | 50 |
| BC, right | ..... | ..... | ..... | ..... | normal | ..... | ..... | ..... | ..... |
| left | | 0 | 50 | 45 | 55 | 50 | 50 | | |

Function of the right equilibrial labyrinths was normal; the left had no function.

On her first visit vision in both eyes was normal with normal pupils, reflexes and fundi. On her return she had coarse nystagmus, Grade 1, on looking to each side. On examination of the fundi, pallor, Grade 1 (on a basis of 1 to 4), of the temporal portion of the right optic disk was found with a suggestion of pallor on the left. She reported having lost the lower portion of the field of both eyes for about two weeks during the preceding summer. Perimetric fields showed enlargement of the blind spots and small arcuate scotomata below them. Although these findings might have been the residua of a retrobulbar neuritis the preceding summer, they suggested the possibility of multiple sclerosis. Roentgenograms of the head showed calcification of the meninges on the right side near the vertex in the parietal area. Roentgenograms of the mastoids were negative. Neurological examination confirmed the presence of complete left facial palsy of a peripheral type. Taste was absent from the anterior two-thirds of the left side of the tongue. The patient also hopped poorly on the left foot. The finger-to-nose test was —1 on the left and the heel-to-knee test was –2 on the left.

A diagnosis of cerebellopontine-angle tumor was made with reservations. The middle ear was exposed by Lempert's procedure of lifting the inferior portion of the drum and replacing it after the biopsy has been taken. The pathologist reported chemodectoma.

A total of 1,200 gamma roentgens was given to the tumor region between March 13 and 15, 1943. Radium was used at a distance of 2.5 cm. Three months later (June, 1943) the patient was given an additional 700 gamma roentgens. The middle ear appeared negative. At this visit the encephalograms revealed internal hydrocephalus with rather marked enlargement of the subarachnoid pathways. Subsequently a diagnosis of multiple sclerosis was made.

Four years later a spinofacial anastomosis was done elsewhere. The surgeon reported no evidence of recurrence of any kind in the ear.

The patient's home doctor stated that when she was last seen by him in 1954, her multiple sclerosis was considerably worse but the chemodectoma had not recurred.

CASE 11.—This patient, a woman aged 49 years, had begun to notice a pulsation in the left ear with some mild aching in the region of the left mastoid three years before admission in January, 1951. In November, 1950, the pain in the left mastoid region became more severe; penicillin did not affect the symptoms.

On examination at the clinic the left tympanic membrane was moderately red and thickened and the landmarks were moderately obscured. The red color appeared to be shining through from the middle ear. A presumptive diagnosis of a neoplasm in the left middle ear was made. Biopsy was refused.

The patient returned in June, 1951. Hearing had decreased in the left ear and she had had some vague dizziness but no vertigo. The left ear appeared unchanged, but a slight haziness involved the entire left mastoid as shown in the roentgenogram. The audiogram showed perceptive loss on the right and a combined deafness on the left.

The left mastoid was explored through a postauricular incision. The entire cellular structure of the mastoid was found to be filled with a thick, brownish fluid and no bleeding was encountered. The middle ear was filled with a bloody exudate which was sucked out. The mucosa appeared normal. Biopsy showed normal mucosa. The external auditory canal was cut across and a Lempert flap fashioned; the operation was completed as a modified radical mastoidectomy because it was felt that the condition was a type of serous otitis media.

By November, 1951, the cavity was completely epithelized and dry. The appearance of the drum was unchanged.

In September, 1953, the patient complained of increased pulsation in the left ear and some discharge from the left external auditory canal. Only a remnant of the ear drum was present and granulationlike tissue was seen in the middle ear. Several biopsies were taken; a final one was reported to be chemodectoma. There was little bleeding from the lesion at any time.

Two courses of roentgen therapy were given. The first, in September, 1953, delivered a tumor dose of 1,450 roentgens and an additional 1,400 roentgens was given in the second course in January, 1954.

The tumor in the middle ear did not change until September, 1954, when it decreased slightly.

CASE 12.—About a year and a half before coming to the Mayo Clinic in August, 1952, the patient noticed gradually increasing deafness in the left ear. A little later a high-pitched ringing tinnitus was referred to the left ear.

On examination the left tympanic membrane was red, distorted and bulging into the canal for about 0.5 cm. The audiogram showed:

| | 125 | 250 | 500 | 1000 | 1500 | 2000 | 3000 | 4000 | 6000 | 8000 |
|---|---|---|---|---|---|---|---|---|---|---|
| AC, right | 20 | 20 | 10 | 20 | 15 | 20 | 20 | 30 | 50 | 50 |
| left | 50 | 50 | 55 | 60 | 60 | 60 | 70 | 80 | 00 | 00 |
| BC, right | | 25 | 25 | 15 | 10 | 10 | 25 | 30 | | |
| left | | 25 | 30 | 20 | 15 | 25 | 55 | 55 | | |

Sufficient masking was used to produce proper lateralization of the hearing.

Roentgenograms of the mastoids showed diffuse clouding of the mastoid cells on the left. The right mastoid was negative.

A diagnosis of chemodectoma was made and verified at biopsy. Operation was done by Lempert's technique of tympanic reflection. External roentgen therapy was given and a tumor dose of 1,080 roentgens was delivered through a single field using 200 kv with an HVL of 0.98 mm of copper.

Eleven months later the left drum, except for a slightly thickened appearance, appeared entirely normal. The audiogram taken at that time showed:

| | 125 | 250 | 500 | 1000 | 2000 | 4000 | 8000 |
|---|---|---|---|---|---|---|---|
| AC, right | 30 | 25 | 15 | 15 | 20 | 30 | 50 |
| left | 10 | 10 | 15 | 25 | 35 | 40 | 55 |

The patient was free of tinnitus or any other symptom in regard to the ear. At present (February, 1955) the patient is symptom free, the tympanic membrane is slightly thickened, there is no evidence of tumor in the middle ear and the gain in hearing has been maintained.

CASE 13.—A woman, 33 years old, consulted us in July, 1947. For two and one-half years deafness had been progressing in the left ear. At first it was intermittent but for more than a year had been constant. A year and a half before admission the patient had an episode of vertigo, nausea and vomiting which was attributed to anemia. Nine months before admission a wart had been found in her left ear canal. Biopsy showed it to be benign and no treatment was given. Two weeks before admission twitching of the muscles of the left side of the face, especially at the angle of the mouth and eyelids, began and a week later she was unable to close the left eyelid tightly.

Examination disclosed that the left external auditory canal was filled with a red, bulging mass. Complete perceptive deafness was found on the left, but function of the equilibrial labyrinth was not tested. Roentgenogram of the mastoids showed destructive changes in the left middle ear, mastoid and petrous pyramid that were probably due to malignancy.

Neurological examination showed only involvement of the VII and VIII cranial nerves on the left.

Biopsy revealed the mass in the right external auditory canal to be a hemangioendothelioma, Grade 1; this was later corrected to chemodectoma.

The right mastoid was explored through a postauricular incision. There was considerable bleeding from the vessels of the mastoid cells. A large tissue mass was removed from the mastoid antrum and middle ear. As the anterior wall of the middle ear was approached, the bleeding became more profuse and careful palpation discovered no bony wall, particularly in the region of the carotid canal, so complete removal of the tumor was not attempted in this area.

Twenty 1-mg radium-element points were packed into the wound and left in place for 30 hours. These were stuck through a strip of gauze which was brought out through the external auditory canal.

After operation the facial palsy on the left was complete; five months later there was nearly complete recovery of function of the facial muscles except the frontalis and the radical cavity was completely epithelized and free of discharge.

A year and a half later a sequestrum was removed from the center of the mastoid cavity. Biopsy was negative for tumor.

In 1950 the patient returned because of a slowly enlarging mass in the right cervical region. This was removed and proved to be a carotid-body tumor.

The ear appeared healed but she had a complete facial palsy on the left and paralysis of the left side of the tongue, the left side of the soft palate and the constrictors on the left, and the left vocal cord was fixed in midposition.

In 1953 a pulsating mass posterior and mesial to the ascending ramus of the left mandible was discovered. Roentgen therapy consisted of a tumor dose of 1,800 roentgens in a six-day period using 250 kv with an HVL of 1.3 mm copper at a distance of 50 cm.

Three months later the mass in the left side of the neck could not be palpated and the pulsation had disappeared. In March, 1954, although the paralysis had persisted, the pulsating mass in the neck was still absent and the mastoid cavity was completely epithelized and free of discharge.

*Pathology.* Within limits, the histologic appearance of chemodectomas is quite constant. The round or polygonal tumor cells, often designated as "epithelioid," usually have abundant slightly granular eosinophilic cytoplasm and round or oval nuclei with punctate granules of chromatin and small, sometimes multiple, nucleoli. The tumor is highly vascular, and the walls of most of the blood vessels consist of little more than a single layer of endothelial cells. The tumor cells lie in ribbons or nests in intimate association with the blood vessels. Trabeculae of connective tissue traverse the tumor, producing a lobulated or alveolar pattern.

The proportion of vascular to epithelioid elements varies; the more vascular tumors may simulate and at times have been diagnosed hemangioma or hemangio-endothelioma.

Most of the tumors in this series exhibited a rather typical structure. In Case 5 the histology was atypical. The biopsy was taken from the tumor presenting in the nasopharynx after it had received intensive irradiation. The tumor cells and their nuclei varied markedly in size and shape. The nuclei were hyperchromatic with coarse chromatin granules. Mitotic figures were numerous. Many of the tumor cells were spindle-shaped and arranged in irregular bundles. The tumor had a markedly anaplastic appearance which led to an original diagnosis of fibrosarcoma, Grade 4. However, on review of the sections a small area was seen in which the cell type and structure typical of chemodectoma were still recognizable.

## REVIEW OF MATERIAL STUDIED

We have reviewed the records of 13 patients with chemodectoma of the glomus jugulare. Since two of these cases had been reported by Figi and Weisman, 11 cases are added to the literature.

*Symptoms and Differential Diagnosis.* In our group of cases the symptoms and signs were nonspecific as was noted by previous observers. Roentgenograms of the mastoid and base of the skull were of little diagnostic or prognostic value. Evidence of widespread destruction of the petrous pyramid was helpful in deciding as to operability, however.

In seven patients (Cases 2, 3 and 5 to 9) the symptoms appear to have been initiated by an acute infection of the upper respiratory tract. In Case 4, the first symptom was discharge from the external auditory canal associated with painful swelling of the ear. In Cases 5, 6 and 8 a chronically discharging ear was present.

It seems possible that the presence of chemodectoma in the middle ear may favor its invasion by pathogenic organisms. It is therefore necessary in a few instances to differentiate chemodectomas from chronic suppurative otitis media.

Pain was an initial symptom present in only two cases.

Tinnitus was one of the initial symptoms in three cases (1, 9 and 12) and loss of hearing was one of the first symptoms in four cases (7, 9, 12 and 13). It is apparent that these symptoms are of no value in suggesting the possibility of chemodectoma in the middle ear or temporal bone.

The symptoms and signs presenting themselves at the initial admission are of more diagnostic interest.

Signs of involvement of a cranial nerve except the VIII are not in themselves suggestive of chemodectoma, but when a primary malignancy of the nasopharynx or a rapidly advancing cancer of the middle ear can be ruled out, the early appearance of a jugular foramen syndrome may be suggestive of chemodectoma, especially if severe pain in the distribution of the V nerve is not present.

A mass in the external auditory canal was found in eight patients (Cases 1 to 6, 8 and 13) and a mass was seen through the drum or producing bulging of an intact drum membrane in five patients (Cases 7, 10 to 13).

The type of a mass in the external auditory canal or middle ear can only be diagnosed by biopsy. We recently saw a patient whom we suspected of having chemodectoma because of a small red tumor behind the membrana tensa. Biopsy proved it to be an adenocarci-

noma, Grade 1. Thus even a tumor appearing to originate in the middle ear and secondarily pushing out the drum is not pathognomonic of chemodectoma, although this lesion is most likely to produce such an appearance.

Excessive bleeding of a mass in the external auditory canal has been frequently mentioned in the literature as a reason for suspecting the presence of chemodectoma. The converse is not true however, because in two of our patients the bleeding from manipulation of the mass in the middle ear or external auditory canal was negligible.

Chemodectoma must usually be distinguished from cancer of the external auditory canal or middle ear, a neurofibroma of the cerebellopontine angle, a neurofibroma of the V cranial nerve in the middle ear or a primary malignancy of the nasopharynx.

*Malignancy of Chemodectoma.* In our cases there was no instance of metastasis and the rate of advance of the lesions varied considerably. In seven patients the lesion had involved other cranial nerves than the auditory, usually the facial.

From the first symptom until the appearance of the palsy there was a period of two and one-half years in Case 1, nine years in Case 2, seven years in Case 5, one and one-quarter years in Case 7, three years in Case 10, seven months in Case 11, and two and one-half years in Case 14.

This variability considered in conjunction with the relatively slow advance of these lesions in all cases, makes assessment of the benefits of any type of treatment somewhat difficult, but the evidence indicates that without treatment or with surgical treatment alone there was in most patients a steady advance in the lesion. This is evidenced by the gross appearance of a tumor mass, involvement of cranial nerves, or increase in the extent of the lesion as demonstrated by roentgenography.

The tendency to produce paralysis of cranial nerves certainly gives evidence of a tendency for these tumors to be malignant by position.

*Effect of Surgery.* In some of our patients chronic suppuration had been added to the tumor. In these instances surgical interference may have benefited the patient.

In no instance is there clear evidence that surgical measures alone prevented the advance of the lesion. In all cases, when operation alone was used, the advance of the tumor seemed to continue unabated until irradiation was used.

*Effect of Radiotherapy.* Of patients who were treated with radiation therapy alone, there was usually little evidence of regression of the tumor. However, in only one instance (Case 8) was there evidence of advance of the lesion after radiation therapy. A tumor dose of 1,230 roentgens was given in 1948 to this patient. In 1951 and 1952 there was evidence of renewed growth and the patient received 1,500 roentgens. One year later progress of the lesion seemed arrested.

The advance of the tumor appeared to have been arrested on observation by the home physician or at the clinic one and one-half years after radiation therapy in Case 1, after one year in Case 9, after ten years in Case 10 and after three years in Case 12.

Both surgical procedures and radiation therapy were employed in eight cases. Symptoms advanced after surgery in Case 2. No increased destruction could be seen in the pyramid but roentgenograms of the skull showed marked destruction of the posterior fossa one year after irradiation. Subjectively the condition was the same about seven years after irradiation.

Patient 3 showed no evidence of recurrence one year after operation and irradiation; patient 4, 15 months afterward; patient 5, 22 years after irradiation and biopsy; patient 6, six months after operation and irradiation and patient 7, six months after irradiation. Patient 11 showed little evidence of effect from 2,850 roentgens of radiation therapy after biopsy. Patient 13 showed evidence of recurrence of chemodectoma six years after operation and treatment with twenty 1-mg radium-element points. However, a tumor dose of 1,800 roentgens caused complete clinical disappearance of the tumor in three months.

When cranial nerves were involved radiation therapy seemed to have little or no effect in restoring function.

*Technique of Radiation Therapy.* In an attempt to control malignant lesions of the middle ear and mastoid with radiation therapy, tumor doses of from 5,000 to 7,000 roentgens and more must be used. For chemodectomas, however, doses in the range of 1,200 to 2,400

roentgens appear sufficient. Such doses may be administered in from one to two weeks, using x-rays generated at 200 to 250 kv with a half-value layer of from 1 to 2 mm of copper. If the tumor is well lateralized, a single portal on the affected side is used. When the tumor is near the midline, directly opposed lateral portals are used to crossfire the tumor-bearing tissue.

The response of chemodectomas to irradiation is slow and the tumor may continue to shrink for several months after treatment. Skin effects from irradiation are minimal. Temporary epilation may occur if hair-bearing tissues are included in the treatment fields.

*Results of Treatment.* It seems reasonable to conclude that surgical treatment has been of little benefit to any of these patients, except those who had secondary chronic purulent infection. Improvement following surgical intervention always seemed to depend on radium or roentgen therapy. In the absence of radiation therapy, lesions of at least four patients progressed and in a few they progressed relatively rapidly.

Definite recession of the lesion followed radiation therapy in four patients in this series.

Evidence of recession in a chemodectoma of the glomus jugulare was recently reported by Huppler and associates.[6] In their case symptoms of dizziness and blacking out on minimal hyperventilation suggested that the tumor might have physiologic function. These abnormal physiologic responses disappeared after roentgen therapy to the tumor.

No further progress was evident in the lesion following radiation therapy in the remainder of our patients so treated.

We believe that it is fair to conclude that radiation therapy has a definite therapeutic effect on chemodectomas of the glomus jugulare. The results seem to indicate that if surgical procedures are used, they should always be combined with radiation therapy.

While too little time has passed in most of the patients who were treated with radiation therapy alone to come to a definite conclusion that radiation therapy alone is to be preferred in most patients with chemodectoma, there are indications that in time this will be the final conclusion.

## SUMMARY AND CONCLUSIONS

Thirteen patients with chemodectoma of the glomus jugulare were treated with a combination of surgical and radiation therapy or with radiation therapy alone. It seems probable that in the future, uncomplicated cases of chemodectoma may be best treated by radiation therapy alone.

## REFERENCES

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man. (Abstr.) Anat. Rec. 79(suppl. 2):28, 1941.

2. Guild, S. R.: The Glomus Jugulare, a Nonchromaffin Paraganglion, in Man. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 62:1045-1071 (Dec.) 1953.

3. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid. Arch. Otol. 41:64-67 (Jan.) 1945.

4. Lattes, Raffaele and Waltner, J. G.: Nonchromaffin Paraganglioma of the Middle Ear (Carotid-Body-Like Tumor; Glomus-Jugulare Tumor). Cancer 2:447-468 (May) 1949.

5. Mulligan, R. M.: Chemodectoma in the Dog. (Abstr.) Am. J. Path. 26:680-681 (July) 1950.

6. Huppler, E. G., McBean, J. B., and Parkhill, Edith M.: Chemodectoma of the Glomus Jugulare: Report of a Case With Vocal Cord Paralysis as a Presenting Finding. Proc. Staff Meet., Mayo Clin. 30:53-58 (Feb. 9) 1955.

7. Dockerty, M. B., Love, J. G., and Patton, M. M.: Nonchromaffin Paraganglioma of the Middle Ear; Report of a Case in Which the Clinical Aspects Were Those of a Brain Tumor. Proc. Staff Meet., May Clin. 26:25-32 (Jan. 17) 1951.

8. Figi, F. A., and Weisman, P. A.: Cancer and Chemodectoma in the Middle Ear and Mastoid. J.A.M.A. 156:1157-1162 (Nov. 20) 1954.

9. Rosenwasser, Harry: Glomus Jugularis Tumor of the Middle Ear (Carotid Body Tumor, Tympanic Body Tumor, Nonchromaffin Paraganglioma). Laryngoscope 62:623-633 (June) 1952.

10. Brown, L. A.: Glomus Jugulare Tumor of the Middle Ear. Clinical Aspects. Laryngoscope 63:281-292 (Apr.) 1953.

11. Riemenschneider, P. A., Hoople, G. D., Brewer, David, Jones, David, and Ecker, Arthur: Roentgenographic Diagnosis of Tumors of the Glomus Jugularis. Am. J. Roentgenol. 69:59-65 (Jan.) 1953.

12. Capps, F. C. W.: Treatment of Glomus Jugulare or Tympanic Body Tumours of the Middle Ear and Petrous Bone With Particular Reference to Radiotherapy. Exerpta Med. [Sec. 11, Otorhinolaryngol.] (Abstr. 1046) 6:181, 1953.

13. Weille, F. W., and Lane, C. S., Jr.: Surgical Problems Involved in the Removal of Glomus-Jugulare Tumors. Laryngoscope 61:448-459 (May) 1951.

14. Barton, R. T., and Thee, E. J., Jr.: Nonchromaffin Paraganglioma: Report of Three Cases. J.A.M.A. 151:619-621 (Feb. 21) 1953.

15. Mattick, W. L., and Burke, E. M.: Glomus Jugulare Tumors. Laryngoscope 62:311-322 (Mar.) 1952.

16. Graf, Kurt: Quoted by Mattick, W. L., and Burke, E. M.[15]

17. Bradley, W. H., and Maxwell, J. H.: Neoplasms of the Middle Ear and Mastoid: Report of Fifty-four Cases. Laryngoscope 64:533-556 (July) 1954.

18. Strong, M. S.: Glomus Jugulare Tumor of the Middle Ear: A Clinical Note. A. M. A. Arch. Otolaryng. 60:145-148 (Aug.) 1954.

LIX

# A REVIEW OF SOME RECENT STUDIES CONCERNED WITH THE EXTERNAL EAR

B. H. Senturia, M.D.

L. H. Sophian, M.D.

S. P. Chiang, Ph.D.

O. H. Lowry, M.D.

St. Louis, Mo.

During World War II the problem of external otitis became an important one to the Armed Forces because of the great loss of time and efficiency of personnel it occasioned. A review revealed little fundamental work had appeared in the recent literature and that otologic therapy in the United States was dominated by the theory that most of the diseases of the external ear were caused by fungus infestation.

A preliminary investigation of the problem was begun at the Air Forces School of Aviation Medicine and the Air Forces continues to provide the major support for this investigative program. Some assistance has been given by the Los Angeles Research Study Club and the St. Louis Otological Foundation.

With this generous help, the following plan of research has evolved (Table I). It was our belief that if the chemical composition of secretions of the normal skin of the ear canal were known, it would be an easy matter to assay the pathological changes and apply the necessary corrective measures.

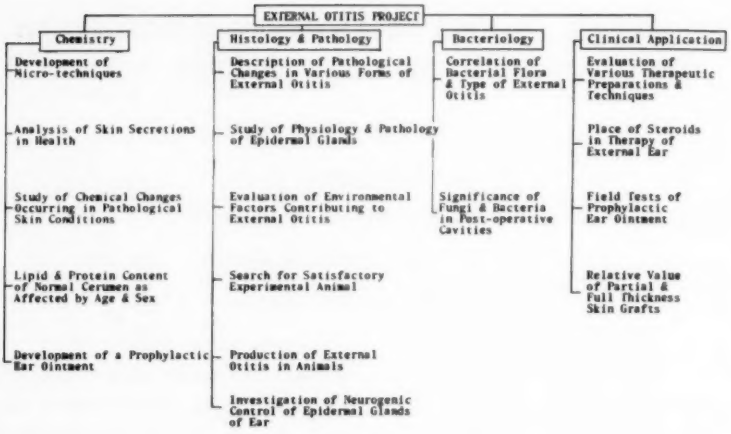
Unfortunately, very little information was found in the literature regarding the chemical constituents of normal ear secretions.[1-3]

---

From the Departments of Otolaryngology and Pharmacology, Washington University School of Medicine, St. Louis.

These studies were performed under Contract AF 33(038)-28643 between the USAF School of Aviation Medicine, Randolph Field, Texas, and Washington University School of Medicine, St. Louis, Missouri, and supported in part by grant from the St. Louis Otological Foundation.

Table I.


EXTERNAL OTITIS PROJECT
Chemistry
Development of Micro-techniques
Analysis of Skin Secretions in Health
Study of Chemical Changes Occurring in Pathological Skin Conditions
Lipid & Protein Content of Normal Cerumen as Affected by Age & Sex
Development of a Prophylactic Ear Ointment
Histology & Pathology
Description of Pathological Changes in Various Forms of External Otitis
Study of Physiology & Pathology of Epidermal Glands
Evaluation of Environmental Factors Contributing to External Otitis
Search for Satisfactory Experimental Animal
Production of External Otitis in Animals
Investigation of Neurogenic Control of Epidermal Glands of Ear
Bacteriology
Correlation of Bacterial Flora & Type of External Otitis
Significance of Fungi & Bacteria in Post-operative Cavities
Clinical Application
Evaluation of Various Therapeutic Preparations & Techniques
Place of Steroids in Therapy of External Ear
Field Tests of Prophylactic Ear Ointment
Relative Value of Partial & Full Thickness Skin Grafts

Some data can be derived from studies of the skin of other parts of the body but we must keep in mind that there are histological differences between these areas, but in the main it was necessary to develop new chemical techniques and procedures for handling the very complex ear secretions.

Table II shows the lipid fractions of skin secretions in per cent of total fat as found in a literature survey and as contrasted with our own findings. A striking similarity between the findings for cerumen and other secretions can be seen. Akobjanoff et al.[4] approached the problem by fractionating cerumen and evolved the following procedures (Table III) for the separation of the free fatty acids. Paper chromatography was then employed in an attempt to separate and identify the individual fatty acids. Figure 1 is a chromatogram which compares various known fatty acids with that of cerumen. Despite intensive efforts to characterize the fatty acid components, this promising approach was found unsatisfactory and had to be discarded. Further efforts in this direction using ultra-violet spectroscopy, polarography and infra-red spectrophotometry were not productive and our efforts were therefore directed to another approach.[5] We are now attempting to perform a complete analysis of

## Table II.

LIPID FRACTIONS OF SKIN SECRETIONS IN PERCENTAGE OF TOTAL FAT

| Secretion | Free Fatty Acids | Triglycerides | Non-Sap. | Cholesterol | Squalene | Paraffin Alcohols, etc. | Phospho-lipids |
|---|---|---|---|---|---|---|---|
| Cerumen | | 54* | 27-42 | 1.8-5.2* | 1-12 | | 0.2-1.1* |
| Sebum (forearm) | 22-32 | 28-41 | 26-38 | 4.4-5.9 | 3.3-14 | 25.0 | 1.1-1.2 |
| Skin lipid (forehead) | 40 | 25-69 | 2-22 | 2.3-6.5 | | 5-43 | 0.0-4.5 |
| Hair fat | 38-63 | 2-20 | 21-37 | 4-13 | 1.4-7.0 | | |
| Sweat lipid (hand) | | 71 | 28 | | 1.4 | | |
| Vernix caseosa | 2-10 | 33-42 | 27-49 | 20 | 2.6 | | 0.03 |

*our own data

## Table III.

**Relative amounts of various lipid fractions obtained from 28.5 gm of cerumen. Reprinted by permission, from Jl. Inv. Dermat. 23:1, July, 1954.**

Cerumen 28.5 Gm.
Alcohol-ether extraction

Alcohol-ether soluble 20.6% (5.88 g.) | Alcohol-ether insoluble

↓

Anhydrous ether extraction

Ether soluble 46% (2.75 g.) Lipids, phospholipids | Ether insoluble Non-lipids 54%

↓

Ice-cold acetone (to precipitate phospholipid)

Acetone soluble Total lipids 94% (2.61 g.) | Acetone insoluble 6% (0.14 g.) Phospholipids

↓

Saponify with alcoholic KOH and extract with petroleum ether

Petroleum ether layer (Non-saponifiable fraction) 39% (1.02 g.)

↓

Cholesterol (free and esterified), other sterols, squalene, waxes, high alcohols

Water layer (Saponifiable fraction)
Acidify with HCl and extract with petroleum ether

↓

Petroleum ether layer 61% (1.47 g.)
Free fatty acids and fatty acids from triglycerides

cerumen. The fact that cerumen is of complex nature and is available only in small quantities necessitated the development of microtechniques for individual samples.[6]

With proper modifications it is now possible to analyze specimens of cerumen as small as 0.2 to 0.3 mg. Samples of the ear wax are smeared onto the center of cleansing tissue, dried under high vacuum at room temperature, and then weighed to the nearest microgram. Total lipids are estimated by the weight loss after extraction. The protein content of the residue is measured using a colorimetric method.

With the use of such microtechniques a total of 120 individual samples of cerumen were examined for lipid and protein content. The results were analyzed according to sex and age and indicate that cerumen obtained from children have slightly higher lipid and lower protein content than adults. No significant difference was found with respect to age among adults nor is there any noted between the sexes.[7]

Such chemical micro-techniques are also being applied to the study of the response of individual patients to therapy. Thus patients with "infantile dermatitis" of the ear have been shown to have gross changes in the lipid and protein fractions of their ear wax. In Figure 2 it may be seen that the lipid content of this patient's ears is far below normal for her age group. The normal findings of her identical twin are an interesting contrast. As these observations are extended, we may be able to offer an explanation for this poorly understood otologic condition and provide the basis for proper therapy.

Let us turn to the next aspect of this program, the histologic and pathologic studies of the external ear. Some excellent investigations are reported in the older literature[8-10] but no correlation of normal and pathologic changes were found. More recently, O'Brien,[11] Shelley,[12] Lobitz,[13] and Sulzberger et al.[14] have concerned themselves with the function and malfunction of epidermal glands, but emphasis has been directed to other areas than the ear.

In planning for the study of the histology of the external ear, it was arranged that all discarded skin from fenestrations and biopsies from still born and autopsies be sent to our laboratory. Such a series of cases was assembled and made possible a study of normal skin of the ear canal.[15]

In the specimens of this control series the lining epithelium of the canal was, as a rule, abundantly keratinized, even in infants. Numerous sebaceous and apocrine glands were present in the cartilaginous meatus but were found only rarely in the osseus portion. The relationship of the apocrine ducts in the adjacent structures was variable. In a count of a hundred apocrine gland ducts which could be followed in the serial sections, 83 opened into pilosebaceous units while 12 were traced to direct openings onto the surface. The rest seemed to have branches suggesting the possibility of multiple exits. These findings were in agreement with Takeda[16] but conflict with those of Alzheimer.[10]

In cross sections of the external auditory canal at all levels, blood vessels and nerves were readily observed and formed a consistent pattern. In the outermost layer, whether in the cartilaginous or bony portions, the arteries subdivided so that three or more groups of arterial vessels were seen at varying distances from each other on the circumference of the canal. Arborization in a circular pattern occurred just outside the deepest of the apocrine glands. In some specimens the appearance was almost comparable to the plexiform network of the nasal mucous membrane and it is therefore referred to as the vascular tunic.

Nerve branches were seen abundantly in the outer layers of the walls of the external auditory canal in all sections examined. Most, but not all, of the fibers were non-medulated. Nerve bundles in many instances ran parallel to blood vessels and approached the glands very closely often giving the appearance of breaking up into fine plexuses which spread out along the basal contractile layers of the gland.

Considerable weight is attached by our group to the richness of the vascular supply and the position of the vascular tunic. The existence of this vascular layer may well affect all types of reactive processes in the lining of the external auditory canal. The severity of congestion in this area is very likely enhanced by the abundance of small thin wall blood vessels. Moreover the location of the tunic within the confines of the cartilages must result in severe tissue tension leading to pain from congestion and compression of nerves. In any area of persistent congestion, edema is prolonged and may lead to atrophy of epithelial elements and an increase in fibrous tissue. These sequences could result whether the congestion arose from inflammatory, allergic or endocrine causes.

Since we wished to chart the changes from the normal to the pathological, it became necessary to obtain biopsies from patients with acute and chronic diffuse external otitis. With adequate antibiotic coverage, specimens were obtained without complication in five patients with acute diffuse external otitis. Serial sections as well as a diagrammatic reproduction of pathologic changes are shown.[17]

In all the sections examined, striking changes were observed in the epidermis. The total thickness of the epidermis was often greatly increased by cellular proliferation and edema. The surface layer showed considerable thickening with irregularly arranged masses of keratinized cells, many of which retained their nuclei. Occasionally the superficial portion of the stratum corneum was lifted free leaving small spaces which contained fluid. Hyperplasia of the prickle cell and basal cell layers often progressed to a striking degree. The cells were increased in number as well as size and inter- and intracellular edema were prominent. The rete pegs were elongated and broadened. The dermato-epidermal margin was edematous and frequently ill defined. Neutrophils could be found infiltrating all the layers of the epidermis and occasionally a micro-abscess was found.

The inflammatory phenomena were greatest in and around the capillary loops in the superficial part of the dermis and consisted of dilatation, engorgement, necrosis, thrombosis and rupture. Vascular changes were also demonstrable in the deeper portions of the corium. Sometimes thrombosis and inflammatory infiltrates were found in vessels closely adjacent to the acini of apocrine glands. There was also a diffuse infiltrate which involved both the superficial and deep corium. In the severest cases foci were massive and consisted of aggregates of neutrophilic leucocytes with liquefaction necrosis. Surrounding them was a dense but diffuse infiltration of lymphocytes, monocytes and a high percentage of eosinophile leucocytes.

The apocrine glands were intimately involved in the inflammatory process. In general, apocrine glands seemed to be atrophic, decreased in size and their epithelium gave the appearance of being non-functioning. There was a diminution of the glandular lumina and for the most part the glomeruli were embedded in the exudate. Leucocytes were sometimes observed within the walls and in some instances in masses within the gland space. The sebaceous glands were often involved in the superficial edema and exudate and poly-

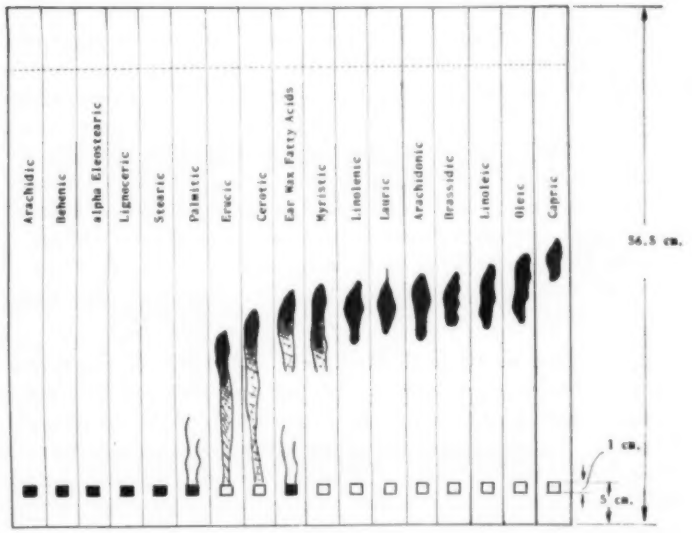



Fig. 1.—PbS spots produced by various pure preparations of fatty acids known to occur in the human body in comparison to those of ear wax fatty acids. Reprinted by permission, from Jour. Inv. Dermat. 23:1, July, 1954.

nuclear leucocytes infiltrated the margin of the glands. The holocrine cells however appeared normal in size and shape.

It is conceivable that compression of the apocrine and sebaceous glands may result from edema and infiltration of the periglandular fibrous tissue. It may be possible that interference with the vascular supply to the glands may be responsible for the degeneration of the epithelium and its apparent lack of function. The absence of dilatation or distension of the acini or ducts seemed to exclude mechanical obstruction of the apo-pilo-sebaceous unit as a cause of the morphologic changes in the apocrine glands although keratotic masses were seen filling the outlets of many of the apo-pilo-sebaceous units.

It is recognized that the eosinophiles found in the dermis suggest an allergic nature in the inflammatory process but this is not in accordance with the morphologic picture observed in acute diffuse

external otitis. Nevertheless there are certain aspects of the picture which may be compatible with a state of sensitization.

The skin of the meatus obtained from 40 patients operated for chronic otitis media and mastoiditis showed many consistent and interesting histopathological changes[18] and provided the opportunity to study the transition from the acute to the chronic stage of external otitis. Our observations revealed the following: All sections of the epidermis showed some thickening of the stratified squamous epithelium. Keratinization was comparable to that observed on exposed skin surfaces. Parakeratosis, intra- and intercellular edema and elongation of the rete pegs were often observed. There was a suggestion of keratin plugs obstructing the orifices of the apo-pilo, sebaceous units.

In the deep portions of the dermis inflammatory exudate gave the appearance of alternating cellularity and fibrosis. It tended to be grouped principally around the accessory glandular structures and their ducts. In such areas the infiltration was relatively compact and surrounded in cup fashion, a network of small blood vessels and lymphatics. Endothelial proliferation and occlusion were commonly observed in the capillaries. Thrombosis of small vessels and attempts at recanalization were evident.

Exudate, consisting mainly of lymphocytes and large monocytes, was seen surrounding the coils of the apocrine glands accompanied by varying degrees of fibrosis. Variable degrees of vacuolization and degeneration were found in the epithelium of these glands. The epithelium was fusiform and the lumen reduced to a crevice. Cystic dilatation of some tubules of an apocrine lobule was often found. The ducts of the apocrine glands were frequently embedded in dense fibrous tissue. Peri- and intraductal infiltration of leucocytes sometimes occurred.

The increased amount of fibrous tissue and vascular engorgement in the superficial part of the cutis seemed to cause little alteration in size or shape of the sebaceous glands. No evidence of secretory obstruction could be detected. In view of the findings on chronic diffuse external otitis described below, the absence of any changes within the sebaceous glands in the acute or chronic otitic studies is noteworthy.

Two skin specimens of chronic diffuse external otitis were obtained. A preliminary review of these slides[19] revealed a remarkable

PERCENTAGE OF LIPID, PROTEIN, AND NON-LIPID NON-PROTEIN

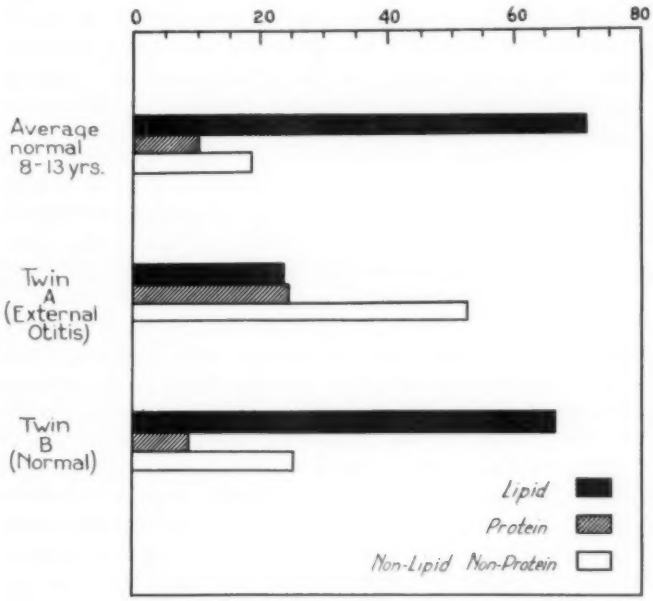



Fig. 2.

absence of sebaceous glands although some intact apocrine glands were found. These observations appear to offer support for the theory that the underlying mechanism in the pathogenesis of diffuse external otitis may be a malfunction of the epidermal glands of the skin of the ear canal.

A second aspect of our pathologic studies is the experimental phase. For some time a satisfactory experimental animal has been sought but the best specimens, the horse, pig and chimpanzee, offer numerous problems in housing and care. We are now utilizing the cat ear which has been shown[20] to have characteristics similar to that of the human. Cats are being exposed to high temperatures and extreme humidity in an attempt to evaluate the importance of envi-

ronmental influences on the secretions of the epidermal glands and on the bacterial flora of the skin of the ear canal.[21,22] It is hoped in this manner to produce controlled infections of the animal ear and thus chart the changes in the lipid and protein content of the secretions. Simultaneously biopsies of the epidermal glands and bacteriological studies will be performed. If this can be achieved it will be possible to evaluate the efficacy of various prophylactic and therapeutic preparations and techniques.

It is our belief that the study of the bacterial flora of the ear canal offers definite but limited opportunities for prevention or cure of diseases of the external ear. Excellent bacteriologic studies have appeared.[23,24] Recently an attempt was made to correlate the bacterial flora and the clinical course of the disease without success.[25] We were impressed, however, with the fact that gram-negative bacilli which were cultured in a high percentage of cases of acute diffuse external otitis indicated superimposed infection and served as reliable evidence of the existence of underlying malfunction of the epidermal glands.

Since most postoperative mastoid cavities are lined with split thickness skin grafts from which the epidermal glands have been cut away, some consideration was given to a possible relationship between the use of this glandless skin and the prevalence of fungi in postoperative mastoid and fenestration cavities. It would appear that recently there has been a rise in the incidence of otomycosis.[23-25] This was particularly evident in our study of the use of hydrocortisone and neomycin in the mastoid and fenestration cavities.[26] Many factors may have contributed to this increased occurrence of fungus infestation but we are impressed with the possibility that the absence of certain vital lipids on the surface of irritated or malfunctioning skin is responsible for the rapid growth of these exogenous organisms.[27]

I should like, briefly, to touch on the clinical phase of these investigations. Based on our concept of pathogenesis of diffuse external otitis and to some extent on our chemical findings, ointments containing some of the vital lipids have been formulated. If this theory has merit, such a prophylactic preparation should prevent superimposed infection in the external ear. We are hopeful that a prophylactic ear ointment will be available shortly for validation.

After infection has occurred active therapy must be instituted. Numerous otic preparations containing one or more antibiotics and

chemotherapeutic agents in oily vehicles have appeared. Recently an attempt to evaluate the relative efficacy of five such formulations was made.[25] In this controlled study, the medical observers had no choice of therapy and progress notes were recorded by independent observers. We were impressed with the lack of superiority of one preparation over another. Of more than passing interest was the observation that fatty acids and saline used as controls were effective in a small number of cases and that one antibiotic dissolved in a simple aqueous vehicle showed as good results as those with combinations of antibiotics, antiseptics and analgesics suspended in a propylene glycol or glycerol vehicle. This latter confirms the findings in a pilot study[28] showing the efficacy of an aqueous vehicle as a carrier of antibiotics in the treatment of acute diffuse external otitis.

Finally, I should like to comment on the role of the various corticoid preparations in the prevention and treatment of external ear diseases. In properly selected categories of external otitis and infections of postmastoidectomy and postfenestration cavities, where there appears to be malfunction of epidermal glands, striking results were obtained with the topical use of various preparations containing hydrocortisone.[29] Thus, in the neurogenic group of ear diseases, combinations of hydrocortisone acetate and neomycin in an ointment vehicle rapidly controlled the disease and eliminated the need for radiation therapy. A similar combination in an aqueous vehicle resulted in the cure of a small number of chronic persistent postoperative mastoid and fenestration otorrheas. The mechanism by which topically applied corticoids produce these changes is not understood but is being studied in relation to the function of the epidermal glands.

We are hopeful that technics and procedures may be developed which will allow us to gain more understanding of the chemical and pathological changes occurring in the skin of the external ear. With a proper appreciation of these fundamental processes, rapid progress in the prophylaxis and treatment of diseases of the external ear can be expected.

500 N. Skinker Blvd.

This coordinated research program would not be possible without the help of many professional and technical associates and assistants. We are particularly indebted to the administrative help of Colonel Paul A. Campbell, Lieutenant-Colonel James E. Lett and the Surgeon-General of the Air Forces.

## REFERENCES

1. Nakashima, S.: Über die Chemische Zusamensetzung des Cerumens, Ztschr. f. Physiol. Chem. 216:105-109, 1933.

2. Zorzoli, G.: Microchem. Research on Some Pigmented Extracts of Human Cerumen, Monit. Zool. Ital. 60, Supple. to Atti Soc. Ital. Anat. 378-82 (1951) C.A. 47,9382i (1953).

3. Sobel, H.: Squalene in Sebum and Sebum-Like Materials, J. Inv. Dermat. 13: 333, 1949.

4. Akobjanoff, L., Carruthers, C., and Senturia, B. H.: The Chemistry of Cerumen: A Preliminary Report, J. Inv. Dermat. 23:1, 43-50, 1954.

5. Lowry, O. H., Rosebrough, N. J., Farr, A. L., and Randall, R. J.: Protein Measurement with the Folin Phenol Reagent, J. Biol. Chem. 193:265, 1951.

6. Chiang, S. P., and Lowry, O. H.: Unpublished data.

7. Chiang, S. P., Lowry, O. H., and Senturia, B. H.: Unpublished data.

8. Von Troltsch, A. F.: Page 9, A Textbook of the Diseases of the Ear, 4th Ed., A. Politzer. Lea Brothers and Co., Philadelphia and New York, 1903.

9. Politzer, A.: A Textbook of the Diseases of the Ear, 6th Ed. Lea Brothers & Co., Philadelphia and New York, 1926.

10. Alzheimer, A.: Über die Ohrenschmalzdrüsen. Verhandl. d. Phys. Med. Wurzburg, bd. 22, 1888.

11. O'Brien, J. P.: A Study of Miliaria Rubra. Topical Anhydrosis and Anhidrotic Asthenia. Brit. J. Dermat. and Syph. 59:125, 1947.

12. Shelley, W. B., and Hurley, H. J., Jr.: Methods of Exploring Human Apocrine Sweat Gland Physiology, A.M.A. Arch. Dermat. and Syphilol. 66:156-161, 1952.

13. Lobitz, W. C., and Campbell, C. J.: Physiology of the Glands of the Human Ear Canal: Preliminary Report Apparatus and Technique. J. Inv. Dermat. 19:2, 125-135 (Aug.) 1952.

14. Sulzberger, M. B., and Herrman, F.: The Clinical Significance of Disturbances in the Delivery of Sweat. Chas. C. Thomas Co., Springfield, Ill., 1954.

15. Sophian, L. H., and Senturia, B. H.: Anatomy and Histology of the External Ear in Relation to the Histogenesis of External Otitis, Laryngoscope 64:9, 772-784 (Sept.) 1954.

16. Takeda, S.: Comparative Histological Examinations of the Skin of the External Auditory Canal, Okajimas Folia Anatomica Aponica 23:295, 1951.

17. Sophian, L. H., and Senturia, B. H.: Pathology of Acute Diffuse External Otitis. Annals of Otology, Rhinology and Laryngology 44:300 (Mar.) 1955.

18. Sophian, L. H., Cooper, Z. K., and Senturia, B. H.: Pathologic Changes of the Skin of the External Auditory Canal in Chronic Otitis Media and Mastoiditis. Annals of Otology, Rhinology and Laryngology 63:2:261 (June) 1954.

19. Liebmann, F. M., and Sophian, L. H.: Chronic Diffuse External Otitis. Unpublished data.

20. Montagna, W.: The Glands in the External Auditory Meatus of the Cat, J. Morph. 85:3, 423-442, 1949.

21. Senturia, B. H.: Diffuse External Otitis: Its Pathology and Treatment, Tr. Amer. Acad. Ophth. and Otolaryng. 147-159 (Nov.-Dec.) 1950.

22. Senturia, B. H.: Diffuse External Otitis: Pathogenesis and Treatment No. 2. To be published.

23. Syverton, J. T., Hess, W. R., Krafchuk, J., Josselyn, E. H., and Freyvogel, G. H., Jr.: Otitis Externa—Clinical Observations and Microbiologic Flora, Arch. Otolaryng. 43:212-225 (Mar.) 1946.

24. Singer, D. E., Freeman, E., Hoffert, W. R., Keys, R. J., Mitchell, R. B., and Hardy, A. V.: Otitis Externa—Bacteriological and Mycological Studies. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 61:317-330 (June) 1952.

25. Senturia, B. H., Cross, R. J., Lett, J. E., and Hardy, A. V.: An Evaluation of Certain Therapeutic Agents and Procedures in the Treatment of Acute Diffuse External Otitis. Laryngoscope 64:12, 1001-1019 (Dec.) 1954.

26. Senturia, B. H., and Alford, V.: Hydrocortisone Acetate and Neomycin in Otic Infections. Laryngoscope 64:10, 834-844 (Oct.) 1954.

27. Senturia, B. H.: Symposium: Medical Management of Chronic Otic Skin Infections. Rationale of Therapy, Tr. Amer. Acad. Ophth. and Otolaryng. (Sept.-Oct.) 1954.

28. Alford, V., and Senturia, B. H.: Unpublished data.

29. Senturia, B. H., Alford, V., and Walsh, T. E.: Topical Hydrocortisone and Neomycin in the External Ear. To be published.

* * *

*(Scientific papers of the American Otological Society to be continued.)*

# The Scientific Papers of the American Broncho-Esophagological Association

## LX

## ELECTROCARDIOGRAPHIC STUDIES DURING BRONCHOSCOPY IN RATED-RISK CARDIAC PATIENTS

### A PRELIMINARY REPORT

WALTER H. MALONEY, M.D.

FRANKLIN C. MASSEY, M.D.

PHILADELPHIA, PA.

Diagnostic and therapeutic bronchoscopy in patients with cardiovascular disease has not kept pace with the rapid advances in cardiovascular surgical techniques. Yet such candidates are faced often with the need for these procedures. Frequently patients are denied the benefits derived from bronchoscopy because of reluctance on the part of the physician to recommend them.

Over the past four years we have had the opportunity to see in consultation 177 patients with some significant degree of cardiovascular disease. On these patients 180 bronchoscopies were performed as either a diagnostic or therapeutic procedure. This does not include the current study.

In the beginning these patients were approached with a certain degree of apprehension and with considerable calculated risk. The need for bronchoscopy was carefully considered and the benefit to be gained weighed against the anticipated risk of cardiac arrest or rapid failure. As our experience increased, with a very low incidence of complications attributable to the bronchoscopy, we were asked to see not only the earlier postoperative complications, but the preoperative diagnostic problems as well. To decrease operative mortality and morbidity, preoperative bronchoscopy to evaluate the associated pulmonary disease received increasing acceptance.

---

Division of Surgery, Department of Laryngology and Broncho-Esophagology; and Division of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania.

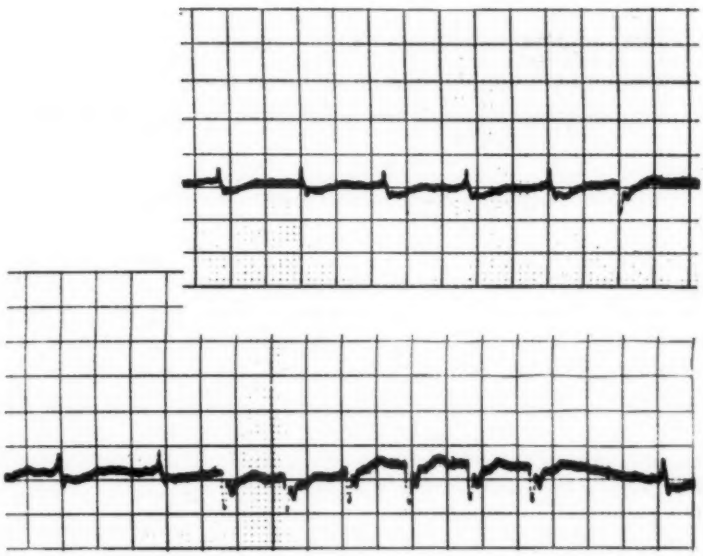


Fig. 1.—Electrocardiograms, A. Case 21—Atrial fibrillation with an occasional ventricular ectopic contraction; B. Same basic rhythm with short runs of ventricular tachycardia.

It became apparent to us that our day by day experience with bronchoscopy in patients with cardiac disease had crystallized the thought that this procedure was not as risky as we had once believed and that the risk was, in fact, over-stressed.

To test the validity of this clinical impression, gained previously by our experience in these 180 bronchoscopies, that indicated bronchoscopy does not materially endanger the welfare of the rated-risk cardiopulmonary patient we studied, as outlined below, the course of 29 such individuals on an otherwise unselected basis.

## MATERIALS AND METHODS

The patients used for this study were ones with clinically significant cardiovascular pathology in whom there was a reasonable suspicion of associated pulmonary pathology as evidenced by productive cough, segmental atelectasis, hemoptysis, wheeze, etc. They were all studied before bronchoscopy from a cardiac viewpoint and a

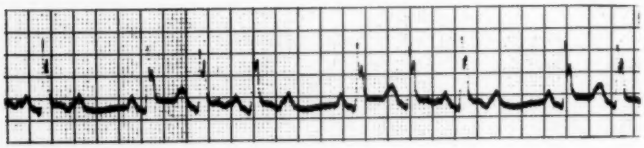


Fig. 2.—Electrocardiogram, Case 25—Second degree heart block.

clinical diagnosis made. Premedication for diagnostic bronchoscopy was usually Demerol,® 50 mg, atropine sulfate, 0.4 mg, by hypodermic, and Nembutal,® 100 mg orally, given one hour before bronchoscopy. The patients were brought to the endoscopic operating room with nasal oxygen being administered and by either wheel chair or stretcher depending on the comfort to the patient. Oxygen, 100 per cent, was administered throughout the whole procedure and the patient returned to his room with it.

The anesthesia for the bronchoscopy was 4 per cent cocaine hydrochloride spray to the pharynx (approximately 1½ to 2 cc vol.), 1 cc of 10 per cent cocaine instilled fractionally into the larynx and 4 cc of 2 per cent xylocaine instilled fractionally into the tracheobronchial tree under mirror guidance.

Following anesthesia, with the patient on the endoscopic table, in bed, or in a wheel chair, prior to bronchoscopy, the following leads were taken using a direct writing Sanborn Cardiette: Leads I, II, III, AVR, AVL, AVF, and Leads V-1 through V-6 inclusive. During the actual procedure of bronchoscopy leads II or V-2 were recorded reverting back to all of the control leads previously mentioned if important abnormalities developed. Particular attention was paid to the record at the times the instrument was introduced into the larynx, at the carina, and each lower lobe bronchus. Following withdrawal of the bronchoscope, additional records were taken before the patient was moved. Technical interference with recording was eliminated by grounding the bronchoscope separately by means of a contact clipped onto it at the handle and attached to the water pipes. The patient and machine were grounded in the usual manner.

Patients done at the bedside as an emergency procedure received only Demerol, 50 mg, and atropine sulfate, grains 1/200, as premedication. The local anesthesia was the same however.

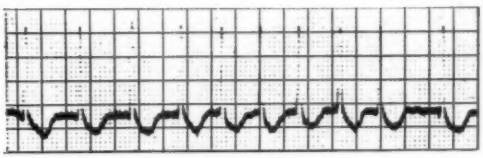


Fig. 3.—Electrocardiogram, Case 26—Atrial fibrillation with moderately rapid ventricular rate.

## RESULTS

Twenty-nine patients were studied in this manner and a total of 32 bronchoscopies done. There was no change in the electrocardiogram in 25 of the bronchoscopies. One had inconsequential changes alone and six had significant changes. One of the latter also had associated inconsequential changes. There were no deaths during bronchoscopy. There were two deaths within a few hours following bronchoscopy, which could be attributed to the procedure or the premedication. Seven additional patients died later, but no connection was made with bronchoscopy as a factor in their demise. The initial rhythm prior to bronchoscopy was extremely varied with major arrhythmias being present in many cases.

## COMMENT

We consider a significant arrhythmia to be one which induces morbidity or mortality because of: 1) its presence only, i.e. ventricular tachycardia, 2) its ability to cause coronary insufficiency, i.e. atrial fibrillation with rapid ventricular rate, or 3) its likelihood of inducing heart failure, i.e. paroxysmal tachycardia.

Changes other than those involving heart rhythm, in the electrocardiogram, have not been discussed here primarily because no significant aberrations (of the ST segment or T wave) were noted in any case. This includes the fact that the position for bronchoscopy varied from flat on the operating table to sitting straight up in a wheel chair (Case 29). From this it seems apparent that hypoxia of the myocardium, induced by coronary insufficiency, is not a pertinent factor in bronchoscopy done in the manner outlined above, stressing the use of oxygen as a supportive measure.

It is interesting to note that in the two cases in which death occurred in a short interval after bronchoscopy there was no change

TABLE I.

DATA ON TWENTY-NINE RATED-RISK CARDIAC PATIENTS UNDERGOING BRONCHOSCOPY

AF - Auricular fibrillation.—ED - Elective diagnostic bronchoscopy.—ET - Emergency therapeutic bronchoscopy.—H.E. - Heart enlarged.—NSR - Normal sinus rhythm.—PVC - Premature ventricular contractions.—VEC - Ventricular ectopic contractions.—Mod.Rap. - Moderately rapid ventricular rate.—LBBB - Left bundle branch block.—Art.scl. - Arteriosclerotic heart disease.—Cor. Pul. - Cor Pulmonale.—Hypert. - Essential hypertension.—Pul.Sten. - Congenital pulmonary stenosis.—Rheum. - Rheumatic heart disease.—TBC - Pulmonary tuberculosis.—RMB - Right main bronchus.—RLL - Right lower lobe bronchus.—LMB - Left main bronchus.—LLL - Left lower lobe bronchus.

| NO. | ETIOLOGY | PROC. | INITIAL RHYTHM | NO CHANGE | SIGNIFICANT CHANGE | INCONSEQUENTIAL CHANGE | ASSOCIATED DISEASE | DIGITALIS | H.E. | SEX AGE | COMMENT |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | Rheum. | ED | AF | x | | | | x | x | F-45 | Death after bronchoscopy |
| 2. | Rheum. | ED | AF | x | | | Bronchiectasis | x | x | M-29 | 1st Br. |
| | | ED | AF,Mod. Rap.,Occ. VEC | x | | | | x | x | | 2nd Br. |
| 3. | Rheum. | ED | AF | x | | | | x | x | F-31 | Death 7 days p.o. |
| 4. | Undet. | ED | NSR | x | | | Bronchiectasis | x | x | F-50 | |
| 5. | Cor.Pul. | ED | Sinus tachy. Occ.PVC | x | | | Emphysema Asthma Tr-bronchitis | x | x | M-49 | |
| 6. | Art.scl. | ED | AF,Mod. Rap.,Occ. VEC | x | | | Old infarct Bronchiectasis Pul. hyperten. | x | | M-66 | Death after bronchoscopy |
| 7. | Hypert. | ED | NSR,Freq. PVC(multifocal) | x | | | Tr-bronchitis | | | M-68 | B.P.-160/110 |
| 8. | Rheum. | ED | AF | x | | | | x | x | F-42 | |
| 9. | Pul.Sten. | ED | NSR,PVC | x | | | TBC | | x | F-23 | Death 14 days p.o. |
| 10. | Rheum. | ED | AF,Mod. Rap.,Occ. VEC | x | | | Supp. lung dis. | x | x | M-42 | Eventual death |
| 11. | Hypert. | ED | NSR | | | Occ.PVC (larynx) | Substernal thyroid | | | F-65 | B.P.-184/100 |
| 12. | Rheum. | ET | Sinus tachy. | x | | | | x | | F-39 | Bedside p.o. |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 13. | Rheum. | ED | AF | x | | | | | x | x | M-53 | |
| 14. | Pul.Sten. | ED | NSR,Occ. PVC | x | | | TBC | | | x | F-24 | |
| 15. | Rheum. | ED | AF,Occ. VEC | | Increased ventricular rate (tongue) | | | | x | x | F-48 | Death 4 days p.o. |
| 16. | Rheum. | ED | AF | | VEC(RLL) Increased ventricular rate (aspirate) | | | | x | x | M-38 | Death 2 days p.o. |
| 17. | Art.scl. | ED | NSR | x | | | Broncho-pneumonia | | x | x | M-50 | |
| 18. | Hypert. | ED | Sinus tachy. | x | | | Bronchitis | | | | M-46 | B.P.-144/110 |
| 19. | Hypert. | ED | NSR | x | | | Tr-bronchitis | | x | x | M-65 | B.P.-194/120 |
| 20. | Art.scl. | ET | NSR,Occ. Premature atrial contrac. | x | | | Acute myocard. infarct, pul. edema, diabetic acidosis, lob. pneumonia | | x | | M-59 | Bedside moribund death |
| 21. | Rheum. | ED | AF,Occ. VEC | | Brief parox. vent. tachycard. (larynx, carina, RMB, LMB, RLL, aspiration) | | Multiple pul. infarcts with abscess | | x | x | F-44 | 1st Br. |
| | | ET | Atrial flutter 2:1 | | PVC (Lar.,LMB) Couple (LLL) | | | | | | | Death 4 days after 2nd Br. |
| 22. | Rheum. | ED | NSR,Occ. PVC | | Few multifocal PVC (aspiration) | | Bilateral Bronchiectasis | | | x | M-63 | Out patient |
| 23. | Rheum. | ED | AF, Mod.Rap. | x | | | | | x | x | F-39 | |
| 24. | Rheum. | ED | AF, Mod.Rap. | | Increased ventricular rate (introduc.) | Occ.VEC | | | x | x | M-49 | |
| 25. | Rheum. | ET | NSR,2nd degree heart blk. | x | | | Post-surg. pericarditis | | | | M-18 | |
| 26. | Rheum. | ED | AF | x | | | | | x | x | F-56 | |
| 27. | Art.scl. | ET | NSR,1st degree heart blk. LBBB | x | | | Supp. lung dis. Chr. cong. failure | | x | | F-79 | |
| 28. | Rheum. | ET | Atrial flutter fibrill. | x | | | Post-surg. pericarditis | | x | x | M-41 | Bedside p.o. |
| 29. | Rheum. | ED | AF | x | | | Supp. lung dis. | | x | x | M-59 | 1st Br. |
| | | ED | AF | x | | | | | x | x | | 2nd Br. |

in the cardiogram during the procedure. In the one case (Case 1) the patient was returned to her room where she was observed to be in pulmonary edema. This was relieved by prompt medical management and was controlled for two hours. She then went into cardiac arrest and died despite every effort at resuscitation. A second patient (Case 6) was over-sedated, was bronchoscoped although lethargic, returned to his bed in the ward where he was found dead two hours later. The impression was that respirations were retarded to the extent that death was primarily one of respiratory depression.

The fact that seven additional patients went on to eventual death indicates in some degree the severity of the disease process in these cases.

There were five patients in whom significant changes occurred during bronchoscopy. All were rheumatic in etiology (Cases 15, 16, 21, 22, 24). Three of the five went on to eventual death.

Inconsequential disturbances of rhythm seem less apt to occur than might be anticipated (Cases 11, 24). If a disturbance of existing rhythm does appear, it is likely to be of a significant nature.

Major arrhythmias existing prior to instrumentation likewise tended to remain unchanged, although it was in this group (particularly with atrial fibrillation) that the significant changes did occur.

In only one instance did an unimportant variant of normal rhythm become transferred into an arrhythmia of major importance (Case 22), giving support to the benignity of bronchoscopy as a diagnostic and therapeutic measure in these cases.

## CONCLUSIONS

From this study it seems to us that the risk of bronchoscopy in patients with significant cardiovascular pathology is over-stressed.

Patients with associated pulmonary disease should not be denied the benefit of bronchoscopy as a diagnostic measure if it is medically indicated. Conventional indications for bronscoscopy are applicable equally to the cardiovascular patient.

Bronchoscopy as a therapeutic measure should be resorted to more frequently and sooner in patients with cardiovascular disease in whom there are postoperative pulmonary complications.

Special caution must be observed in cases with major arrhythmias because if these tend to be altered upon instrumentation the variation is likely to be significant. It would seem advisable to do these cases

with electrocardiogram assistance and with a physician in attendance who is interested in, acquainted with and able to treat medically the major arrhythmias if necessary.

We feel that a more accurate evaluation of these findings will be apparent as the series is enlarged and that out of such a series can come a clearer definition of the place of bronchoscopy in the management of cardiovascular disease problems. We hope, also, this preliminary report will stimulate others into similar studies and analyses of their experience.

230 NORTH BROAD ST.

We wish to thank George D. Geckeler, M.D., Clinical Professor of Medicine and Cardiology, for his helpfulness and valuable suggestions in facilitating these studies.

REFERENCES

1. Maloney, W. H.: Peroral Endoscopy in Cardiac Surgery, Tex. State Jour. of Med. 49:861-864 (Dec.) 1953.

2. Massey, F. C.: Clinical Cardiology, Williams and Wilkins, Baltimore, 1953.

3. Pullen, R. L.: Pulmonary Diseases, Lea and Febiger, Philadelphia, 1955.

LXI

# OBSERVATIONS ON AQUEOUS AND OILY MEDIA IN LUNGS OF EXPERIMENTAL ANIMALS

L. C. Smith, M.D.

AND

James A. Harrill, M.D.

Winston-Salem, N. C.

Iodized oils were used as early as 1904 and were introduced as contrast media for bronchography in 1922 by Sicard and Forestier.[3,7] Early experimental work, using cats, showed phagocytosis to be the prime factor in removing the oil from the tracheobronchial tree. There are certain objections to the use of iodized oils as contrast media in bronchography, such as, lipoid pneumonia, iodized oil granuloma, insolubility of the oils in the bronchial secretions, and its slow removal from the lungs with interference in subsequent radiographic studies.

Several water soluble preparations are in use now for bronchography. In general, these consist of iodopyracet (3,5 diiodopyrodone-n-acetic acid diethanolamine) and a viscosifying agent in the form of a cellulose derivative. The iodopyracet is known in America as diodrast, in Sweden as umbradil and in Switzerland as ioduron.[11] The viscosifying substance, carboxymethyl cellulose, is a cellulose ether made by the reaction of monochloracetic acid with alkali cellulose. The sodium salt is soluble in water. It is removed from the lungs by phagocytosis, ciliary action, and cough. The iodopyracet component is excreted through the kidneys. Other substances (pectin, acacia, dextran and others) have been tried as viscosifying agents but were found to be less desirable.[9]

There are three main water soluble substances now in use for bronchography—methocel-diodrast, America; umbradil viscous B, Sweden; and ioduron B, Switzerland.[11]

In 1948, Morales and Heiwinkel[9] of Stockholm did experimental work with umbradil viscous B and found that it could be given to dogs orally and intravenously without adverse effect. The dogs were injected intravenously daily for three months with a total dose of 20 grams being given. They also injected it intra-articularly and subcutaneously in calves without adverse effect. When used in a small

series of humans for bronchography it produced less cough than did iodized oils. In subsequent work with humans, Morales and others concluded that it was more irritating than iodized oils and to obtain the best results the patients should be positioned with an anesthetic agent prior to introducing the umbradil viscous B into the tracheobronchial tree.

In 1949, Hellström and Holmgren[4] injected umbradil viscous B through a catheter into one lobe of the lungs of white rats and rabbits. They found that most of the animals showed a normal picture in 48 hours. Saline was used as a control and produced hyperemia of the lungs.

Atwell and Pedersen,[1] in 1950, found that they obtained better results when the carboxymethyl cellulose was increased from 2.6 per cent to 3.3 per cent. This diminished the alveolar spread.

About the same time Morales began working with umbradil viscous B, Vischer[5] began work on ioduron B, which is a similar but a more viscous preparation. He found histologically demonstrable traces of the carboxymethyl cellulose four months after injection. The reaction in the lungs consisted of thickened septa, decreased air content and accumulations of phagocytes and eosinophils. In surgical and autopsy material in humans a foreign body type reaction with giant cells was observed six to ninety days after bronchography. Other observers disagreed with these findings. Zollinger, as quoted by Hellström, thought that the foreign body reaction was probably produced by accumulation of epithelial mucus. He found that carboxymethyl cellulose could be distinguished from epithelial mucus by using toluidine blue and Hotchkiss periodite stains. Toluidine blue stains both and Hotchkiss periodate will stain mucus and not carboxymethyl cellulose.

Various authors have reported great differences in the results obtained in human and animal lungs between umbradil viscous B and ioduron B. Hellström[5] attempted, using white rats, to compare the results of the two substances under the same conditions. The animals were killed at intervals from two days to six months. After 48 hours most of the umbradil viscous B had disappeared from the bronchioles and alveolar lumina. There was an increase in thickness of the alveolar septa without obvious congestion. At one month, two of six animals showed umbradil in the phagocytes. No foreign body granulomata were seen. Of the group injected with ioduron B, five of six animals killed at six months showed changes with a large amount of contrast material still present.

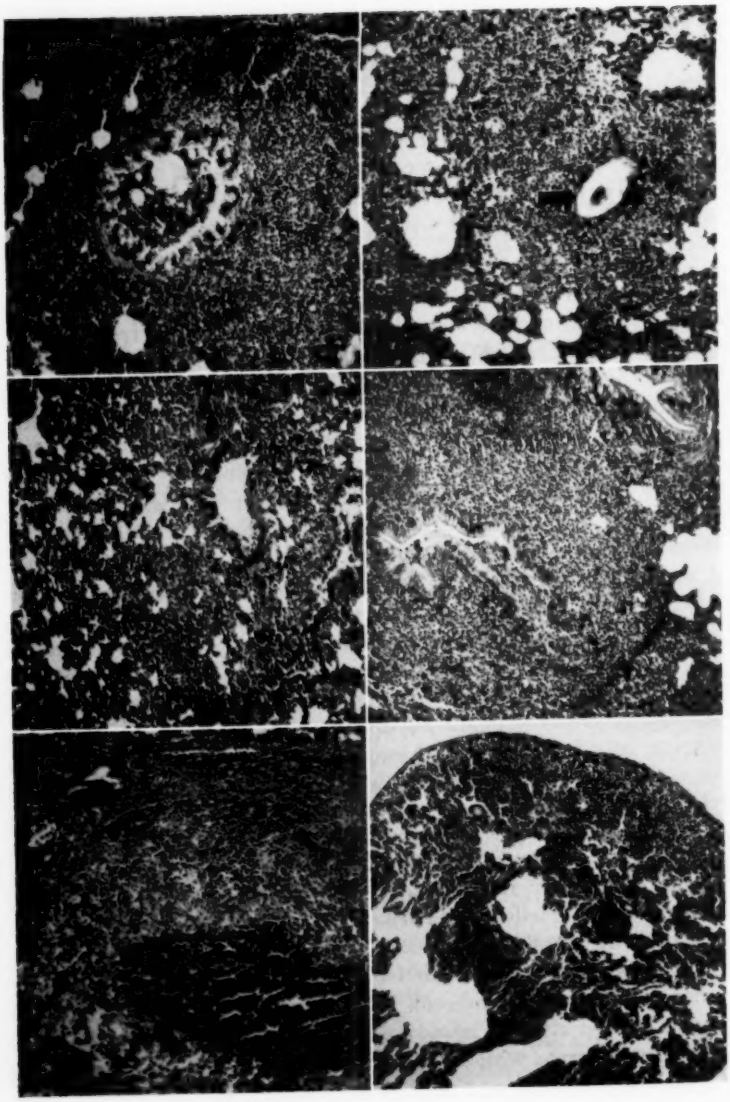


Fig. 1.—Section from rabbit lung, 24 hr. after injection with umbradil viscous B, showing an acute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 2.—Section from rabbit lung, 48 hr. after injection with umbradil viscous B, showing an acute pneumonitis around a globule of carboxmethyl cellulose. Hematoxylin and eosin, X100.

Fig. 3.—Section from rabbit lung, 4 days after injection with umbradil viscous B, showing a subacute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 4.—Section from rabbit lung, 8 days after injection with umbradil viscous B, showing a subacute bronchopneumonia with proliferation of the epithelium lining a bronchiole. Hematoxylin and eosin, X100.

Fig. 5.—Section from rabbit lung, 8 days after injection with umbradil viscous B, showing an area of necrosis surrounded by round cells and macrophages. Hematoxylin and eosin, X100.

Fig. 6.—Section from rabbit lung, 8 days after injection with umbradil viscous B, showing a chronic inflammatory reaction and a foreign body type giant cell. Hematoxylin and eosin, X100.

In general, umbradil viscous B gives a mucosal pattern. Ioduron B is more viscous than umbradil viscous B and fills the bronchi and bronchioles.

## EXPERIMENTAL WORK

The purpose of our experiments was to observe first hand what happens early in the lung of experimental animals when they are injected with umbradil viscous B and to compare this reaction with that produced by iodochlorol and saline. In doing this, three groups of animals were used.

Group I consisted of 14 white rabbits weighing from five to nine pounds. Six of the rabbits were injected intra-tracheally with 1 cc of umbradil viscous B. through an 18 gauge needle without anesthesia. Six others were injected in a similar manner with 1 cc of iodochlorol. The remaining two rabbits were injected with 1 cc of normal saline. All the animals were held upright for one minute to allow the material to flow into the bronchi and bronchioles.

Group II consisted of 15 cats weighing 3.5 to 6 pounds. This group was divided 6, 6, and 3 and injected with 1 cc of umbradil viscous B, iodochloral, and saline respectively, in a similar manner to the rabbits in Group I.

Group III consisted of 17 rabbits weighing 5 to 6 pounds. This group was divided 6, 6, and 5 and injected with 1 cc of umbradil viscous B, iodochloral, and saline respectively. These were also injected without anesthesia.

The animals in each group were killed at intervals of one, two, four, six, eight, and ten days or until all the animals in each series in each group were killed. The animals in Group I were killed by a heavy blow on the head. The ones in Group II and III were killed by an overdose of barbiturate given intraperitoneally. The cats in Group II were found to have chronic pulmonary disease at autopsy and hence will not be included in this report.

## GROSS FINDINGS

The findings in Group I and III were similar and will be described together. The rabbits injected with umbradil viscous B showed patchy areas of reddish-brown discoloration with the lung in these areas being more firm than the surrounding tissue in 24 hours In general, these same findings persisted for ten days, but were progressively less marked after 24 hours.

The animals injected with iodochloral showed changes within 24 hours similar to those injected with umbradil viscous B.

The ones injected with saline showed very little grossly in 24 hours but in 48 hours there was some patchy reddish-brown discoloration. After 48 hours there were no gross changes.

### MICROSCOPIC FINDINGS

The animals killed 24 hours after injection with umbradil viscous B showed patchy areas of bronchopneumonia. Many of the small bronchi and bronchioles were filled with an acute inflammatory exudate and surrounded by areas of pneumonitis with the exudate being composed mainly of neutrophils. In 48 hours the reaction was still pronounced. There was proliferation of the cells lining the alveoli and the exudate contained many large phagocytes with foamy cytoplasm. Globules of carboxylmethyl cellulose were scattered throughout the areas of inflammation in the 24 and 48 hour specimens. After four days the reaction became less acute with the findings consisting mainly of thickening of the alveolar septa, minimal to moderate infiltration of round cells and neutrophils, and many large phagocytes. The 6, 8, and 10 day specimens showed a fairly marked reaction, but the reaction was progressively less acute. In one eight-day specimen there was an area of focal necrosis surrounded by a chronic inflammatory response. Special stains revealed carboxymethyl cellulose in specimens up to 10 days. (Figs. 1, 9)

The sections of the lungs taken from the animals injected with iodochloral showed patchy areas of bronchopneumonia with an inflammatory exudate in many of the bronchi and bronchioles. In the 24 hour specimens the reaction was less acute than that in the 24 hour specimens from animals injected with umbradil viscous B. There was thickening of the alveolar septa with many large phagocytes and minimal to moderate infiltration of neutrophils and round cells. The reaction became more and more pronounced through the sixth day. There were areas of focal necrosis in the tenth day specimens. (Figs. 10, 15)

The animals sacrificed 24 hours after being injected with saline showed a patchy bronchopneumonia with an acute inflammatory exudate. The 48 hours specimens showed a more marked but similar reaction. The fourth and sixth day specimens showed marked improvement with an almost normal picture at six days. (Figs. 15, 19)

Special fat stains were used on some of the specimens injected with iodochlorol. Many globules of lipoid material were demonstrated in the tissue. Sections taken from the lungs injected with

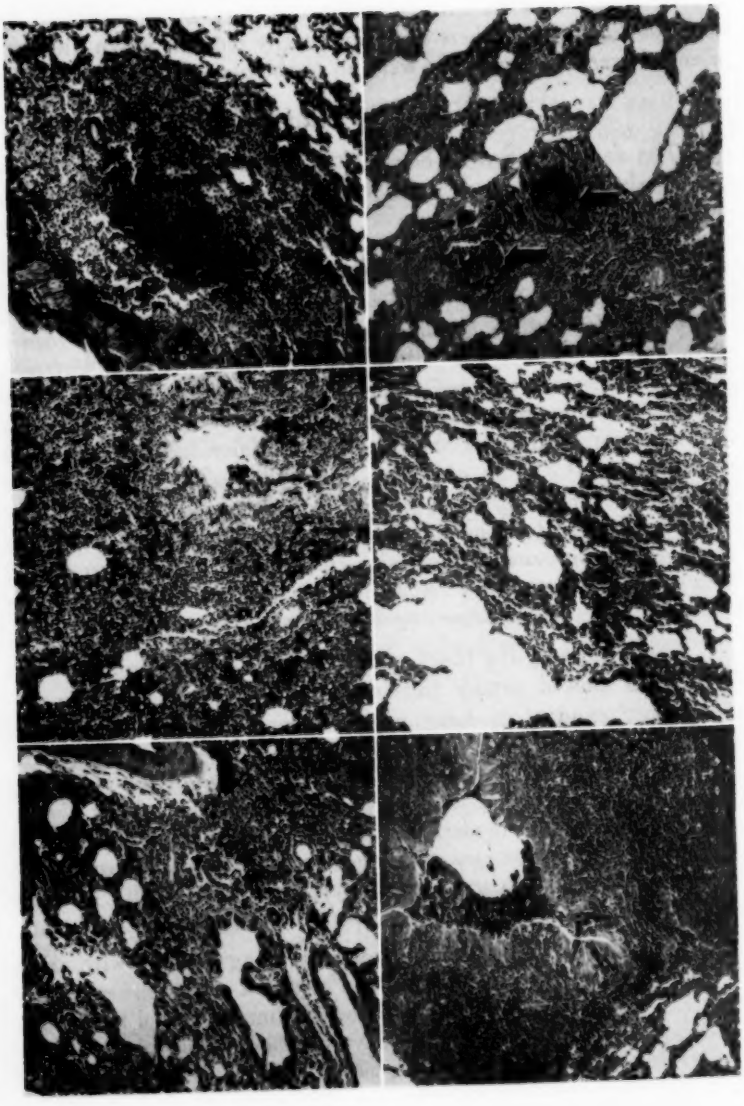


Fig. 7.—Section from rabbit lung, 10 days after injection with umbradil viscous B, showing a small area of necrosis surrounded by a chronic inflammatory reaction containing many large macrophages. Hematoxylin and eosin. X100.

Fig. 8.—Section from rabbit lung, 10 days after injection with umbradil viscous B, showing globules of carboxymethyl cellulose surrounded by marked inflammatory reaction. Toluidine blue, X100.

Fig. 9.—Section from rabbit lung, 24 hr. after injection with iodochlorol, showing an acute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 10.—Section from rabbit lung, 48 hr. after injection with iodochlorol, showing a far less severe reaction than that shown in Fig. 9. There is thickening of the alveolar septa with minimal infiltration of neutrophils. Hematoxylin and eosin, X100.

Fig. 11.—Section from rabbit lung, 4 days after injection with iodochlorol, showing a moderately acute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 12.—Section from rabbit lung, 6 days after injection with iodochlorol, showing a marked bronchopneumonia. Hematoxylin and eosin, X100.

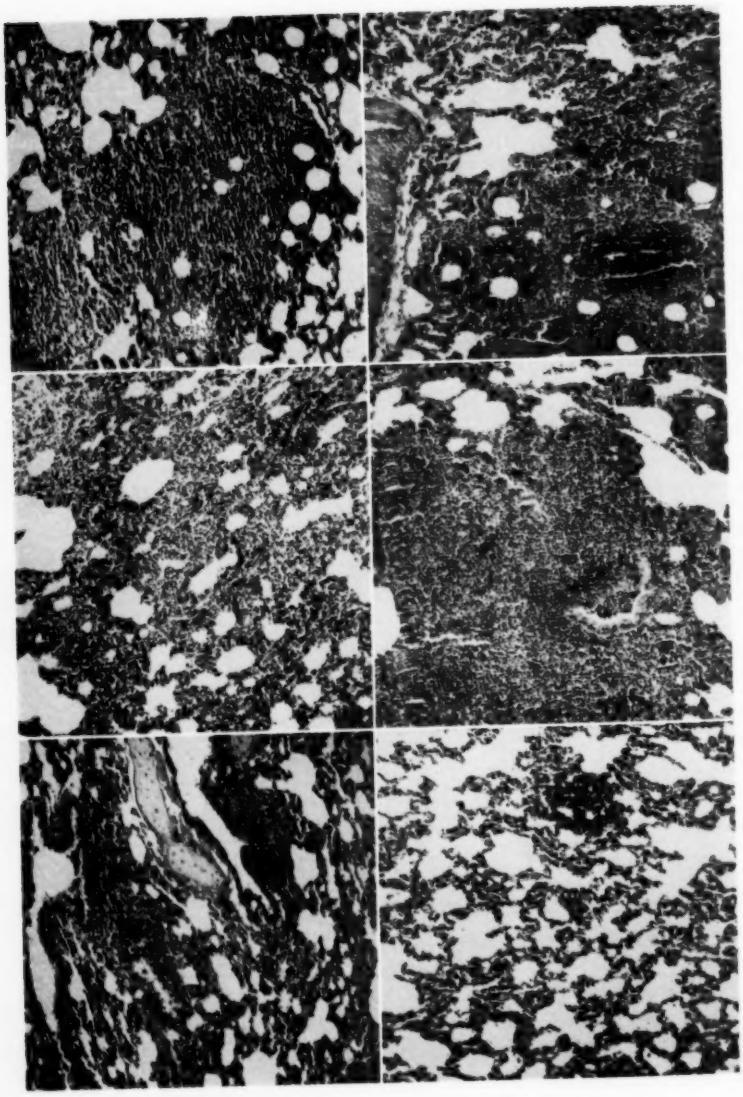


Fig. 13.—Section from rabbit lung, 8 days after injection with iodochlorol, showing a subacute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 14.—Section from rabbit lung, 10 days after injection with iodochlorol, showing a small area of necrosis surrounded by many macrophages, round cells, and neutrophils. Hematoxylin and eosin, X100.

Fig. 15.—Section from rabbit lung, 24 hr. after injection with saline, showing an acute bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 16.—Section from rabbit lung, 48 hr. after injection with saline, showing a marked bronchopneumonia. Hematoxylin and eosin, X100.

Fig. 17.—Section from rabbit lung, 4 days after injection with saline, showing thickened alveolar septa with minimal infiltration of round cells and neutrophils. Hematoxylin and eosin, X100.

Fig. 18.—Section from rabbit lung, 6 days after injection with saline, showing relatively normal lung. Hematoxylin and eosin, X100.

umbradil viscous B were stained with toluidine blue and Hotchkiss periodate. Carboxymethyl cellulose was demonstrated in all the sections up to ten days, the full course of the experiment.


## SUMMARY

A brief incomplete review of the literature has been presented on the use of water soluble contrast media in bronchography. Three main ones are in use—umbradil viscous B, ioduron B, and methocel-diodrast.

Experiments were done to compare the reaction produced in the lungs of white rabbits between a water soluble preparation, umbradil viscous B, and an oily preparation, iodochlorol. Saline was used as a control. The animals were sacrificed at intervals up to ten days. All three substances produced an acute patchy bronchopneumonia. This reaction persisted throughout the course of the experiment with umbradil viscous B and iodochlorol. The animals injected with saline showed a marked reaction in 48 hours with marked clearing within six days.


BOWMAN GRAY SCHOOL OF MEDICINE.

## REFERENCES


1. Atwell, Robert J., and Pedersen, Robert L.: A Water Soluble Contrast Medium for Bronchography, Dis. Chest 18:535, 1950.

2. Brandenstein, Luise C.: Ioduron B Bronchography, Am. J. Roentgenol. 66: 769, 1951.

3. Brown, A. Lincoln: The Fate of Iodized Oil (Lipiodol) in the Lungs, Surg., Gyn., and Obst. 46:597, 1928.

4. Hellström, B., and Holmgren, Hj.: The Reaction of the Lungs on Bronchography with Viscous Umbradil (Umbradil Viskos B) (Astra), Umbradil (Astra), and Carboxymethyl Cellulose. Acta Radiol. 32:471, 1949.

5. Hellström, B.: The Reaction of the Lung on Bronchography with Water Soluble Contrast Media in Rats, Acta Radiol. 40:371, 1953.

6. Hentel, Wm.: Cohen, M. B., and Brandenstein, L. C.: Evaluation of Ioduron B in Bronchography, Dis. Chest 21:280, 1952.

7. Norris, Charles M., and Stauffer, Herbert M.: Aqueous Contrast Media in Bronchography, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 60:802, 1951.

8. Lenzi, Mario: Elimination of the Water Soluble Contrast Medium in Bronchography—Functional Bronchography, Acta Radiol. 37:103, 1952.

9. Morales, O., and Heiwinkel, H.: A Viscous Water Soluble Contrast Preparation, Acta Radiol. 30:257, 1948.

10. Morales, Olallo: Further Studies with Viscous Umbradil (Umbradil Viskos), Acta Radiol. 32:317, 1949.

11. Salzman, Emanuel: Peck, Mordant E.; and Neerken, A. J.: Methocel-Diodrast: A Viscous Water-Soluble Contrast Medium for Bronchography, Radiology 58: 209, 1952.

LXII

# ESOPHAGEAL VARICES IN HEPATIC CIRRHOSIS: AN ENDOSCOPIC STUDY

STANTON A. FRIEDBERG, M.D.

CHICAGO

HUGH D. BENNETT, M.D.

HOUSTON, TEXAS

HARKISHEN SINGH, M.D.

AND

JOHN LINDBERG, M.D.

HINES, ILLINOIS

The aim of this paper is to record the esophagoscopic observations on 674 patients with liver disease, with particular emphasis upon the problem of esophageal varices. Such related aspects as x-ray studies, postsurgical and post mortem findings and incidental endoscopic issues are also included in the analysis.

The opportunity of studying a large series of patients with liver disease stems from the creation of a Liver-Spleen Board at Hines Hospital in 1950. A total of 935 individuals comprise the series thus far (January, 1955), but we have used, for this investigation, only the records of 674 patients with hepatomegaly and abnormal liver function tests in whom the results of both the x-ray and endoscopic search for esophageal varices are available. A coding system, established by the medical service in 1950, has enhanced the accumulation and accessibility of data and the present study is based upon experiences covered by the period 1950 through 1954.

Evaluation of the liver studies in these patients has been a particular long-term interest of Drs. Bennett and Lindberg. The rotation of personnel in a hospital engaged in an extensive resident training program has created more of a problem for the departments of radiology and otolaryngology where 948 esophagrams and approximately 850 esophagoscopies respectively were done on these 674 patients.

---

From the Department of Otolaryngology and the Department of Medicine, Veterans Administration Hospital, Hines, Illinois.

Observations from these latter departments, though not as uniform as if a single representative had assumed sole responsibility throughout, may be regarded as indicative of a fair average of expectant accuracy.

The endoscopic procedures were performed following preliminary medication and, but for rare exceptions, with topical anesthesia alone. Because of the poor barbiturate tolerance of patients with cirrhosis, it was the practice to see and evaluate each candidate for esophagoscopy prior to ordering medication in order to individualize the drug dosages. (The ¾ grain capsules of pentobarbital sodium and secobarbital sodium are particularly useful in this connection.)

The instruments used were the Jackson and Jesberg esophagoscopes. In teaching the techniques of esophagoscopy we have always emphasized the use of filiform bougies as "lumen finders" for men beginning their training.

TABLE I

DIAGNOSIS OF VARICES BY ESOPHAGOSCOPY

| | NO. WITH VARICES | PER CENT |
|---|---|---|
| 674 patients in series (hepatomegaly, abnormal liver function) | 184 | 27.3 |
| 582 patients (cirrhosis) | 175 | 30.0 |
| Transient varices | 16 | 8.7 |

There were 558 patients with Laennec's cirrhosis and 72 patients with fat infiltration of the liver (precirrhotic). In addition, there were 10 patients with hepatomegaly of undetermined cause, 7 patients with hepatoma, 6 with postnecrotic cirrhosis, 5 with portal hypertension of undetermined cause, 5 with obstructive cirrhosis, 3 with chronic hepatitis, 3 with cirrhosis of undetermined cause, 2 with cardiac cirrhosis, 2 with no liver diagnosis, and 1 with hemachromatosis. Hence there were 582 patients with some form of cirrhosis of the liver.

### ESOPHAGOSCOPIC DIAGNOSIS OF VARICES

In the 674 patients with hepatomegaly and abnormal liver function, the diagnosis of esophageal varices was made endoscopically on 184, or 27.3 per cent. If we include only the 582 patients with a diagnosis of cirrhosis, however, varices were noted endoscopically in 175 or 30 per cent. (Table I)

A history of melena, hematemesis or both was obtained in 192 (28.4 per cent) of the 674 patients in the entire series. Of these 192 patients with a history of bleeding, 94 or 49 per cent were found to have varices on esophagoscopy. Of the 184 patients in whom varices were noted on esophagoscopy 94, or 51 per cent, gave a history of melena, hematemesis or both. (Table II)

TABLE II
INCIDENCE OF BLEEDING

| | NO. PTS. | % |
|---|---|---|
| 674 patients in series; history of melena, hematemesis, or bot'h | 162 | 28.4 |
| | VARICES | % |
| 192 patients with history of melena, hematemesis, or both | 94 | 49 |
| | NO. PTS. | % |
| 184 patients with varices — history of bleeding | 94 | 51 |

In the only other endoscopic study of which we are aware, Brick and Palmer reported an incidence of 63.3 per cent of varices in a group of 150 patients with cirrhosis of the liver.[1] It should be mentioned that their observations were made using four-diameter telescopic magnification and that 40 per cent of the patients examined had a history of hemorrhage.

One of the interesting developments in this study is contained in a previous report by Bennett and associates on the phenomenon of transient esophageal varices.[2] In the series to date there have been 16 patients out of 184 or 8.7 per cent, who exhibited transient varices (Table I); precautions were taken to obtain agreement of opinion both on x-ray and esophagoscopy or upon esophagoscopy on two separate occasions by different observers. Palmer and Brick subsequently drew similar conclusions when, on the second esophagoscopy in 60 patients with portal cirrhosis, there was spontaneous disappearance of varices in 7 instances or 8.5 per cent.[3]

On the basis of our observations and those of Palmer it may be estimated that varices noted endoscopically may be absent on subsequent examination in almost 10 per cent of patients. The reverse situation probably also holds. This conclusion, coupled with the intermittent episodes of bleeding in cirrhotic patients, is indicative of variations in portal hypertension not yet fully understood.[4]

In surveying available information on the incidence of esophageal varices in patients with hepatic cirrhosis, one is impressed by the tremendous variation in reported figures.

It is apparent that necropsy studies on patients with cirrhosis who have died of massive hemorrhage will reveal a high incidence of varices, probably around 80 per cent. Attention has also been directed towards other sources of severe bleeding in patients ill with cirrhosis.[5]

In contrast, the incidence of varices in clinical studies on patients who have bled or in routine autopsy studies on cirrhotic patients appears to be somewhat lower, between 50 and 60 per cent.[6,7] (It is well-recognized that meticulous care is required to demonstrate esophageal varices at autopsy and that they may be overlooked unless special techniques for demonstration are utilized.) In addition, varices may be discovered at autopsy on patients without any history of bleeding.

## COMMENT

As a result of this particular investigation we have found that esophagoscopy on patients with cirrhosis of the liver will be positive for varices in approximately 30 per cent of cases. It is conceivable that the incidence of varices noted endoscopically might be even lower than 30 per cent if esophagoscopy were to be done routinely on all patients with cirrhosis. In patients with a history of bleeding, varices were found on esophagoscopy in 49 per cent. Since the presence or absence of varices represents information of considerable value from a prognostic point of view and is of great importance to the surgeon, esophagoscopy is deemed a necessary adjunct in evaluation of the patient with cirrhosis.

## X-RAY DIAGNOSIS OF VARICES

In the 674 patients who were esophagoscoped, x-ray evidence of esophageal varices was reported in 140 patients, an incidence of 20.7 per cent. If we consider only the 582 patients with cirrhosis we find that positive x-ray evidence of varices was noted in 138 or 23.7 per cent.

A positive diagnosis of varices by both endoscopy and x-ray was made in 107 patients or 15.8 per cent.

A positive diagnosis of varices was made by endoscopy in 77 patients (11.4 per cent) in whom the diagnosis was not confirmed by x-ray.

There were 32 patients (4.7 per cent) with x-ray diagnoses of varices not confirrmed by esophagoscopy.

There were 458 patients (67.8 per cent) in whom no varices were noted on either esophagoscopy or x-ray.

If we analyze the figures which represent corroboration of endoscopic and x-ray diagnoses we find that agreement between the two methods occurred in 83.7 per cent of the total number of patients examined. (Table III)

TABLE III

RESULTS OF ENDOSCOPIC AND X-RAY DIAGNOSIS OF VARICES

| | PATIENTS | PER CENT | |
|---|---|---|---|
| Positive diagnosis by both esophagoscopy and x-ray | 107 | 15.8 | |
| Negative diagnosis by both esophagoscopy and x-ray | 458 | 67.9 | |
| | | | 83.7 |
| Positive diagnosis by esophagoscopy unconfirmed by x-ray | 77 | 11.4 | |
| Positive diagnosis by x-ray unconfirmed by esophagoscopy | 32 | 4.7 | |
| | | | 16.1 |
| | 674 | | 99.8 |

(In a previous report Kirsh et al. have listed the total figures which represent agreement and disagreement on both positive and negative endoscopic and x-ray findings in over 500 patients in this same series.[8] Their conclusion was that the endoscopic and x-ray reports were in agreement in 90 per cent of all 502 cases.)

Reports in the literature again vary widely in regard to the demonstrability of esophageal varices by x-ray. On the one hand there is a statement to the effect that "the diagnosis of this important lesion has become established with definiteness so that it is now possible to recognize this lesion prior to operation and without the need of resorting to esophagoscopy or gastroscopy."[9] In contrast, other authors feel that x-ray evidence will be positive in but from 12 to 20 per cent of patients with varices.[10,1] These differences probably reflect the individual interests of the authors in roentgenography, since special techniques are required for the demonstration of varices.

## COMMENT

From the results of this study it would seem that the diagnosis of esophageal varices by x-ray can be anticipated in almost 25 per cent of patients with cirrhosis, providing considerable attention and effort is directed towards working out a satisfactory roentgenographic routine. Standard esophageal barium studies are inadequate for the demonstration of esophageal varices.

The diagnosis of esophageal varices in this series has been made more often by esophagoscopy than by x-ray. We recognize that neither method is infallible but it is probable that there are more variables associated with the filming and interpretation of roentgenograms than with direct visualization of the esophagus. It may be that accuracy of x-ray diagnosis can be increased by further efforts along lines of technique.

### INCIDENTAL ENDOSCOPIC FINDINGS

In the preparation of patients for esophagoscopy we have insisted on vocal cord examination and the instillation of small amounts of local anesthetic into the trachea to minimize cough resulting from pressure of the esophagoscope against the membranous posterior tracheal wall. As a result of laryngeal inspection one patient was observed with extensive leukoplakia of the vocal cords and another individual was found to have an early vocal cord carcinoma.

There were 18 diagnoses of hiatal hernia made on x-ray and esophagoscopy and 11 diverticula of the traction type were seen. The diagnosis of esophagitis was made on 6 occasions and hypertrophic folds of esophageal mucosa were encountered twice.

One patient had an incomplete thin web at the esophagogastric junction and this was cautiously ruptured by passing the esophagoscope into the stomach.

Perhaps the most interesting incidental discovery was that of an early carcinoma of the midesophagus. This had not been seen on x-ray and had not produced any narrowing of the lumen. A positive biopsy was obtained, the esophagus was resected and the lesion was found to be less than 1 cm in diameter. Unfortunately the patient died one year later of metastases to the liver.

### COMPLICATIONS OF ESOPHAGOSCOPY

In over 850 esophagoscopies reported here there were two instances of perforation and both patients recovered, one with surgical drainage, the other with medical treatment only. There was one death in the series which must be indirectly attributed to esophagoscopy. The patient had apparently recovered from an attack of pneumonia prior to endoscopy but following the procedure he developed dysphagia associated with edema of the arytenoids. The pneumonia reappeared and although there was no evidence of perforation or mediastinitis, the pulmonary infection precipitated a liver coma from which he expired thirteen days after esophagoscopy.

There were two other cases where trauma was suspected but the patients had no ill effects.

In only one patient in the entire series was there any bleeding from varices during esophagoscopy. This was of mild degree and was readily controlled by tamponade. One other patient had melena after esophagoscopy. These experiences warrant the definite conclusion that the hazards of bleeding during esophagoscopy on patients with varices are negligible, providing the usual endoscopic safeguards are observed.

Esophagoscopy was designated as "unsuccessful" in 15 cases. These are obviously not included in this study.

### POST MORTEM OBSERVATIONS

As previously recorded, there were 184 patients with liver disease in whom the diagnosis of esophageal varices was made endoscopically. Although no intensive follow-up program has been established, 59 of these patients have died, 23 of whom expired from hemorrhage. Higgins[11] has reported that 59 out of 115 patients studied (51 per cent) died of hemorrhage.

Of the 59 deaths, autopsy examinations were done in 29 patients. Among the 29 autopsies esophageal varices were found in 23, or 79 per cent, in spite of the fact that no specialized techniques were employed for their demonstration. This would lend support to the contentions that the presence of varices carries a poor prognosis[12] and that in patients dying of hemorrhage, varices are found in a high percentage of cases.

Of some interest, but of less significance, are the figures pertaining to the diagnosis of varices by x-ray but not proven endoscopically. In this group of 33 patients 5 have died, none of hemorrhage. There were 4 autopsies and in only 1 were varices noted.

Finally there is a group of 458 patients in whom no esophageal varices were observed on esophagoscopy or x-ray. There have been 20 deaths, 9 from hemorrhage. Among the 13 autopsies, varices were found in 6. The outlook for those patients with liver disease without demonstrable varices is thus considerably more favorable, though a certain percentage of these individuals will eventually develop varices. The sparse figures available in this group probably do not warrant any conclusions.

### POSTSURGICAL OBSERVATIONS

Recording of the results of various types of shunt procedures performed on patients in this group does not fall within the scope

of this report. In some instances the optimal follow-up studies were not obtainable. However, there were 17 determinate patients on whom esophagoscopy was done after surgery. All of these patients had varices proven preoperatively. After the shunt operation 11 of the 17 were observed to have persistent varices. Varices were absent in the other 6 patients. In several of the patients esophagoscopy was done on three to six occasions with identical results.

Esophagoscopy must be considered necessary in the postsurgical evaluation of patients undergoing procedures for the alleviation of portal hypertension.

## SUMMARY

The results of an endoscopic study of 674 patients with hepatomegaly and abnormal liver function are presented.

There were 582 patients with hepatic cirrhosis. In this group, esophageal varices were diagnosed endoscopically on 175 patients or 30 per cent.

In patients with a history of melena, hematemesis or both, 49 per cent were found to have varices on esophagoscopy.

The diagnosis of esophageal varices has been made more often endoscopically than by x-ray in this series. Corroboration of diagnoses by endoscopy and x-ray has been possible in over 80 per cent of the total patients studied.

The hazards of bleeding during esophagoscopy on patients with varices were found to be minimal.

The presence of esophageal varices is of sufficient importance from both prognostic and surgical aspects to warrant routine esophagoscopy on all patients with hepatic cirrhosis.

122 S. Michigan Ave.

## REFERENCES

1. Brick, I. B., and Palmer, E. D.: Incidence and Diagnosis of Esophageal Varices in Cirrhosis of the Liver, Gastroenterol. 25:378-384 (Nov.) 1953.

2. Bennett, H. D., Lorentzen, C., and Baker, L. A.: Transient Esophageal Varices in Hepatic Cirrhosis, A.M.A. Arch. Int. Med. 92:507-522 (Oct.) 1953.

3. Palmer, E. D., and Brick, I. B.: Portal Cirrhosis: Correlation Between the Severity of Esophageal Varices and Variations in Physical Findings, Am. J. Med. Sci., 227:149-151 (Feb.) 1954.

4. Whipple, A. O.: The Problem of Portal Hypertension in Relation to the Hepatosplenopathies, Ann. Surg. 122:449-475 (Oct.) 1945.

5. Fainer, D. C., and Halsted, J. A.: Sources of Upper Alimentary Tract Hemorrhage in Cirrhosis of the Liver, J. A. M. A. 157:413-414 (Jan. 29) 1955.

6. Weinberg, T.: Observations on the Occurrence of Varices of the Esophagus in Routine Autopsy Material, Am. J. Clin. Path. 19:554-557 (June) 1949.

7. Bjornboe, M., and Raaschou, F.: Pathology of Subchronic Atrophy of the Liver, Arch. Int. Med. 84:933-957 (Dec.) 1949.

8. Kirsh, I. E., Blackwell, C. C., and Bennett, H. D.: Roentgen Diagnosis of Esophageal Varices (in press).

9. Ritvo, M., and Shauffer, I. A.: Gastrointestinal X-Ray Diagnosis, Philadelphia, Lea and Febiger, 1952, p. 108.

10. Hare, H. F., Silveus, E., and Ruoff, F. A.: Esophageal and Gastric Varices, with Report of a Case, S. Clin. N. America, 28:729-732 (June) 1948.

11. Higgins, W. H.: The Esophageal Varix: Report of 115 Cases, Am. J. Med. Sci., 214:436-441 (Oct.) 1947.

12. Blakemore, A. H.: Portacaval Anastomosis, Surg. Gyn. and Obst., 87:277-279 (Sept.) 1948.

* * *

*(Scientific papers of the American Broncho-Esophagological Association to be continued.)*

# Clinical Notes

## LXIII

## SYPHILIS AND ORAL CANCER

S. Gordon Castigliano, M.D.
Philadelphia, Pa.

In recent years an increasing interest in cancer has been developing in an ever-growing segment of that group of physicians who limit their energies to diseases which involve the ear, nose and throat. The oral cavity, however, has thus far won little attention from otolaryngologists. In fact it is not rare that such cases are referred by them to dentists. This is unfortunate since the oral cavity is an important site of cancer. Oral malignancy is important, not so much because the attack rate is great, but because the disease once present is so rapidly fatal. It is unquestionably true that the action of the first physician to be consulted by the cancer patient may be the most important link in the chain of events which may lead to ultimate cure or death. This premise being true with respect to general cancer, it is no less true for the patient with oral cancer for it is one of the most rapidly lethal of all cancers (Fig. 1). Thus it is fundamental that correct and decisive action be taken at the earliest moment.

In eighteen short, fast-moving months eighty of one hundred patients with untreated or neglected oral cancer are dead. This means that those early weeks in the career of an oral cancer lesion are precious ones for the patient who is afflicted. What is done or left undone by the physician once he is consulted will nearly always determine the eventual outcome. Prompt, affirmative professional action will give the patient the best chance for salvage. Unhappily, there is evidence to show that a high incidence of professional delay persists which contributes importantly to the low salvage rate of oral malignancy.

Strangely, the oral cavity is a medical "no man's land." The general practitioner in dentistry and the oral surgeon are more concerned with the hard structures of the oral cavity; and the otorhinolaryngologist has been more concerned with diseases of the ear, nose, tonsils, sinuses, the mastoids and larynx. It must be asserted that a certain minimum standard of diagnostic competence in oral cancer is to be expected from the members of a specialty which should claim the oral cavity as part of its rightful domain.

With alarming frequency, mouth cancer is being confused with other conditions which occur in the oral cavity. This confusion has

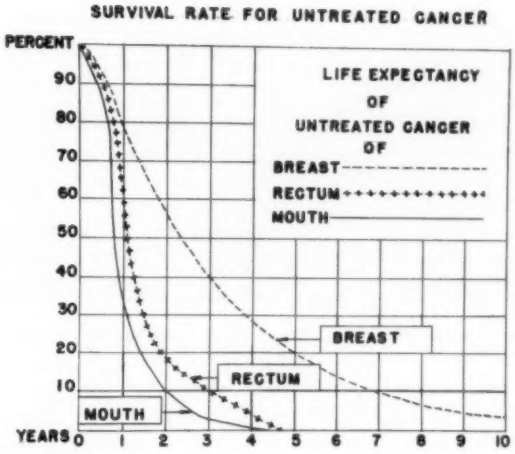



Fig. 1.

been found to be present among those members of the profession best qualified to judge. It should be understood that in no condition with which cancer can be confused is the speedy establishment of the correct diagnosis so vital as it is with cancer. A physician cannot play the waiting game. In many illnesses the accepted medical approach has been to "treat expectantly." Various trials of medication of one type or another are leisurely tested in the hope of striking upon the most useful combination. In oral cancer such an easy-going and languid approach cannot be condoned; it can lead only to the death of the patient. Consequently, it must follow that any disease of the oral cavity which, by any stretch of the imagination, could be conceived to be cancer should be so regarded until disproved by microscopic examination following a correctly performed biopsy. Furthermore it should be pointed out that no contraindication exists to the performance of biopsy. With such an approach, a trial of so-called specific therapy of one kind or another, still practiced by many, will cease.

Syphilis is perhaps the most common disease with which cancer is generally confused, based on the records of the head and neck service of the American Oncologic Hospital. The mystery is why this is so. This contribution deals briefly with this problem.

In the first place, the earlier syphilitic lesions such as extragenital facial or oral chancre and mucous patches are rarely encountered in

Fig. 2.—Mucous patch involving infra-aspect of anterior tongue. (Courtesy National Cancer Institute.)

Fig. 3.—Extragenital chancres in female. (Courtesy New England Journal of Med. and Dr. Walter F. Lever.)

Fig. 4.—Extragenital chancre. Hard chancre of vermillion zone of upper lip. (Courtesy of National Cancer Institute.)

Fig. 5.—Interstitial syphilitic glossitis with associated atrophy, gummatous breakdown and scarring. A rare case of pure tertiary oral syphilis without malignancy.

a head and neck tumor clinic (Figs. 2, 3, 4). This is quite understandable inasmuch as patients who suffer from syphilis in the primary or secondary stage generally are youthful rather than in the advanced middle-age group where the majority of oral cancer is found. This being so, the younger syphilitic patients, quite painfully aware of the diagnostic probabilities, choose their physicians with admirable judgment and hence generally find their way to the dermatologist or the syphilologist.

The extragenital chancre which has been often confused with cancer is the so-called hard chancre most frequently encountered on the lip area, tongue and tonsil (Fig. 4). These lesions too are found usually in youthful subjects acutely suspicious of the diagnosis. These patients usually do not gravitate to the tumor clinic, the oncologist, or the otolaryngologist.

According to the records of the American Oncologic Hospital tertiary syphilis, usually a disease of middle age, is perhaps the most common disease with which oral cancer is confused (Figs. 5 and 6).

Tertiary syphilis then, for all practical purposes, is the only form of syphilis which is seen involving the oropharyngeal structures in the busy head and neck tumor clinic. *But almost always when evidences of tertiary syphilitis are present in the oral cavity, the patient has an associated cancer!* For example, in a survey of 2250 cases of oral malignancy only nine cases of pure tertiary syphilis were encountered. That is to say, only nine cases of tertiary syphilis were found which did not have associated malignant disease involving the mouth. Only one case of pure syphilis, then, is seen to involve the oral cavity at the American Oncologic Hospital to every 250 cases of oral malignancy (Figs. 5 and 6).

Although pure oral syphilis is rare it is important to recognize that syphilis is frequently found to complicate oral malignancy, particularly cancer of the tongue. Nearly thirty per cent of tongue cancer cases have a positive Wassermann. This figure is about six to nine times the average incidence of syphilis in the white male population of this country. Many of these positive Wassermann cases show oral stigmata of syphilis along with cancer. Syphilis is even more commonly associated with oral cancer in certain countries of the Orient.

Thus it can be seen that it is most unwise and dangerous to think of uncomplicated tertiary syphilis as the complete diagnosis when confronted with a patient presenting an oral lesion and a positive serological test for syphilis (Figs. 7 and 8).

TABLE 1.
INCIDENCE OF POSITIVE WASSERMAN IN ORAL MALIGNANCY.

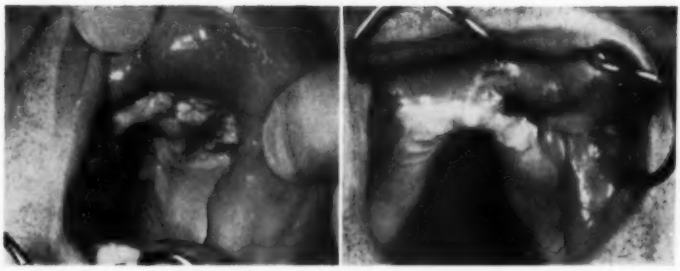
(American Oncologic Hospital)

| SITE | PER CENT |
|---|---|
| Tongue | 29 |
| Buccal Surface | 14 |
| Lip | 9 |
| Gum | 7 |
| Floor | 6 |

Just why some physicians persist in making a diagnosis which is limited to syphilis alone when confronted with a patient presenting an oral lesion associated with a positive Wassermann is difficult to rationalize in consideration of the cold statistics which deal with incidence.

Often the referring physician, undoubtedly, lacks familiarity with the facts just stated. Such a physician will comment, knowingly, that he decided "to spare the patient and give him a chance" with a therapeutic test for syphilis before referring him (belatedly) and "condemning him to the rigorous and useless treatment" of cancer. This statement is almost unbelievable.

In reviewing the histories of oral cancer patients at the American Oncologic Hospital, the basis for such a decision can be found. There are still too many physicians who in the face of an oral lesion incorrectly interpret the positive blood Wassermann reaction or other serological tests for syphilis. Also encountered are members of the defeatist group who erroneously insist that all cancer is completely incurable, therefore, the patient is better served by a course of temporization and deception. It must be emphasized that these specific tests were developed with the intention of diagnosing syphilis with a rather high degree of accuracy. It was never intended that these tests should be used to exclude cancer as a diagnostic possibility. These tests have nothing whatever to do with the diagnosis of cancer. Nevertheless, there are physicians today who, on discovering an ulcerative lesion in the mouth of a patient, persist in obtaining a serologic test for syphilis *before even considering a biopsy*. If the report on the test for syphilis is returned as positive, a therapeutic trial with antiluetic therapy is indulged in "to give the patient a chance." (sic) The



A B

Fig. 6.—A. Gumma involving upper right gingivo-buccal sulcus. Appearance after biopsy. No evidence of malignancy. B. Appearance of same lesion three weeks after intensive penicillin therapy.

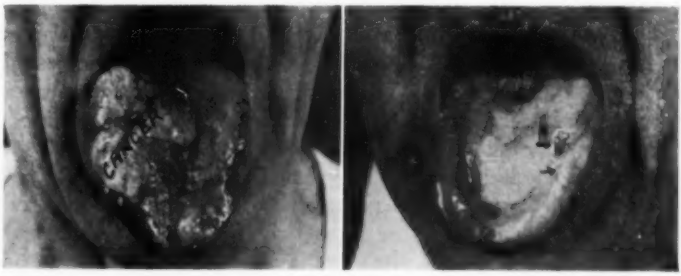


Fig. 7.—Patient with syphilis, interstitial glossitis, leukoplakia, and proliferating squamous cell carcinoma. Patient was treated for syphilis for six months before being referred for adequate management.

Fig. 8.—Patient with syphilis, atrophic glossitis, leukoplakia and malignant degeneration is shown at A.

biopsy is often not done. It must be emphasized again that those physicians who follow this practice are apparently unaware of the odds against them. As a consequence, precious weeks are lost as the cancer inexorably continues to grow, infiltrate and perchance reach a hopeless state before adequate treatment is even suggested.

Frequently patients treated with antibiotics do appear to improve as the mouth in general and associated infection appear to clear up. The treating physician with his first diagnostic impression of syphilis strengthened by this apparent improvement does not give up easily. In our files are examples of cases which develop hopeless metastasis while the treating physician is still evaluating his specific antiluetic therapy. Sadly enough this doctrine of the therapeutic test is still suggested as a recommended procedure by many physicians. Such physicians fail to realize that whatever chance the patient may have will be greatly reduced and too often will be lost by unwarranted delay in establishing the diagnosis of cancer by means of biopsy.

Oral cancer, as is true with any rapidly lethal malignant lesion, should come to be regarded as an emergency disease. Delay in arranging for adequate life-saving management should not be permitted.

Recent writings in texts, cancer manuals, etc., indicate that we have not yet reached the point where cancer is truly looked upon as an emergency disease even by physicians in top level policy making positions. For example, a recent well-known volume dealing with malignancy of the head and neck states that the appearance of syphilitic glossitis is typical. The description of the appearance of such a tongue goes on to mention the thickened and scarred tongue, the atrophy of the papillae and patches of associated leukoplakia. Mentioned also is the lumpy form of syphilitic glossitis apparently resulting from healed gummatous processes. Another paragraph dealing with syphilitic glossitis further states that if *after* two or three weeks of antisyphilitic treatment, the gumma does not heal, a biopsy should be done. Cancer manuals of rather recent publication also read in the same vein.

This is but one of the many indications that physicians are still practicing and teaching the waiting game. I should like to emphasize again that this type of teaching is unsound. It encourages delay.

The appearance of an oral malignant lesion and its early recognition may be influenced strongly by the presence of an antecedent lesion. Occasionally, the antecedent lesion dominates the picture so completely that the unwary or careless observer may be ensnared and thereby may fail to recognize the presence of carcinoma even

when in an advanced stage. Tertiary syphilis can wait two or three weeks or much longer for therapy, but the reverse is not true—cancer cannot wait. Those few weeks may spell the difference between success and failure.

The patient suffering from suspected syphilitic glossitis, gumma, leukoplakia, etc., should have immediate biopsy. Associated cancer must always be suspected at the outset in a patient with oral syphilis.

A single negative biopsy does not rule out cancer in such cases. Multiple biopsies are necessary before a negative report can be relied upon.

Little good will be done if we, by lay education, induce prospective patients to present themselves earlier for examination if we, the physicians, do nothing but carry on in the same old way.

Because of the fact that patients are presenting themselves earlier for examination, a higher index of suspicion is essential. Nothing will have been gained if a cancer-conscious public consults poorly informed physicians. It is therefore fundamental that the general level of professional proficiency in the diagnosis and management of oral cancer be elevated, particularly in that segment of the medical profession best qualified to act.

## SUMMARY

Oral malignancy is a rapidly fatal disease. Eighty of one hundred untreated or maltreated patients are dead in eighteen short months.

The incidence of syphilis in oral malignancy is high — almost thirty per cent of tongue cancer patients have a positive Wassermann.

Pure syphilis of the oral cavity, on the other hand, is rare. At the Oncologic Hospital only one case of uncomplicated oral syphilis (syphilis without oral cancer) is found to every 250 cases of oral cancer.

In the light of these facts the so-called "therapeutic test" for syphilis with alleged specific therapy in Wassermann-positive cases before consideration of biopsy is an unsound and unwarranted procedure.

It should be universally appreciated that in no condition with which oral cancer can be confused is speedy establishment of the correct diagnosis so vital to success as it is with oral cancer.

Oral malignancy as is true with any malignancy should come to be regarded as an emergency disease. Delay in arranging for

adequate life-saving efforts should not be condoned. This is especially true in patients with complicating syphilis since cancer in such cases is even more malignant than the average.

## CONCLUSION

Educators are still far from the achievement of unity of thought concerning the supreme importance of avoiding delay in ruling out malignancy of suspected lesions in the various body areas. On one hand, zealous, resourceful and articulate workers have achieved a remarkably standardized and decisive approach to the early recognition of cancer in some body areas; whereas, in other body areas, much is left to be done before comparable progress is made. Oral malignancy is such a field.

POWELTON AVE. AT 33RD ST.

LXIV

# THE EFFECT OF SALICYLATES ON THE INNER EAR

JULES G. WALTNER, M.D.

NEW YORK, N. Y.

The toxic influence of salicylates on the ear and its clinical manifestations have been known for a long time. The pathogenesis, however, is far from being understood, except for some scattered information.

Tinnitus, hearing loss and vertigo, all three or just one of these symptoms, may be present in salicylate poisoning. The degree of symptoms varies a great deal and there seems to be no direct parallelism between dosage and intensity of cochleo-vestibular disorder. Pronounced toxic symptoms may occur after ingestion of very small doses of salicylates. The pathogenesis of this is unknown. The considerable variation of dosage from person to person, which is necessary to produce symptoms, could be explained by special sensitivity of the patient to this drug, but this does not help us in understanding the pathogenesis of this disorder.

The second moot question of even greater theoretical and practical value is related to the anatomical localization of the lesion in the ear.

Covell's[1] experimental studies on salicylate poisoning revealed mitochondrial changes in the cells of the stria vascularis and of the external hair cells of Corti's organ. The spiral ganglia were unaffected. He found the lower half of the cochlea to be more susceptible to the toxic action of salicylates. He assumed, that the damage to the mitrochondria is due to the direct action of the drug. He considered salicylates a direct protoplasmic poison. He found the blood vessels of the stria vascularis dilated, therefore ischemia was considered unimportant in producing lesions in the inner ear.

Lurie[2] found marked degenerative changes in the cells of the spiral ganglion of experimental animals. Degeneration was most pronounced in the basal turn of the cochlea, but he stated that the

---

From the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University and the Presbyterian Hospital, New York. This study was assisted by grants from the Research Council of the American Otological Society.

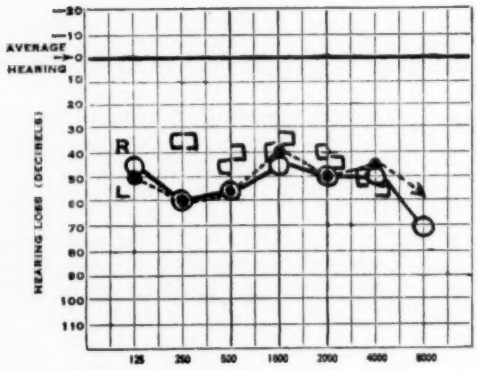



Fig. 1.—Audiogram, on seventh postoperative day, of patient who ingested 200 tablets of aspirin during the first six days following tonsillectomy.

apical turn may be equally involved if administration of salicylates was continued for a longer period of time.

Falbe-Hansen's[3] experimental studies are very interesting from several points of view. His histological findings essentially correspond to what Wittmaack described as "hypertonic degeneration" in the cochlear duct. These findings led Falbe-Hansen to the formulation of the following theory: increased secretion of labyrinthine fluids, especially of the perilymph, is brought about by salicylates and quinine. This gives rise to increased intralabyrinthine pressure with pressure strain exerted on the fenestrae. The increased pressure would explain the aural symptoms including hearing loss.

Intralabyrinthine hemorrhages were found in animal experiments by several authors and these were interpreted as the result of salicylate poisoning (Lurie, etc.). Labyrinthine hemorrhages, however, are not infrequent in post mortem fixation even in the absence of salicylate poisoning, in the experience of this author.

It is evident from the above that there is no unanimity of opinion or evidence as to the histopathology and pathogenesis of salicylate poisoning.

## REPORT OF A CASE

A case of salicylate poisoning is presented, which has many features of a laboratory experiment on a human, and therefore is unique.

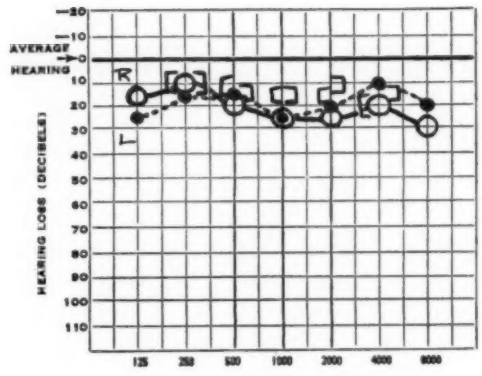



Fig. 2.—Audiogram, ninth postoperative day.

A 28 year old colored woman had undergone a tonsillectomy for recurrent peritonsillar abscesses. During the first six days following operation this patient took 200 tablets of aspirin for what she called "terrific pains." On the seventh postoperative day she complained of hearing loss, recurrent tinnitus, and recurrent sensation of unsteadiness. Examination revealed normal eardrums. Audiometric studies showed bilateral, symmetrical, severe hearing loss both for air and bone conduction and evidence of recruitment (Fig. 1).

Aspirin medication was stopped and the hearing showed considerable improvement two days later (Fig. 2). The patient was aware of the improved hearing and the tinnitus ceased altogether. Vestibular tests performed the same day (Hallpike) showed no evidence of vestibular disturbance. Prothrombin time was 17.5 seconds, within normal limits. This was quite a surprise in view of the large doses of ingested aspirin. Audiometric studies revealed normal hearing seven days after the first hearing test (Fig. 3).

## DISCUSSION

The case throws additional light on the pathogenesis of salicylate poisoning, raising new questions about the histopathology of this disorder in particular and in general, about our usual terminology of inner ear deafness.

The severe loss of hearing both by air and bone conduction and the presence of recruitment definitely qualify the hearing loss in this case as typical nerve deafness. Salicylates may act as direct proto-

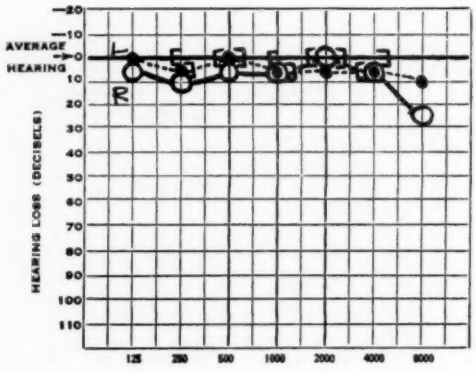



Fig. 3.—Audiogram, sixteenth postoperative day.

plasmic poison, may cause interference with the normal blood supply of the inner ear or may increase the intralabyrinthine pressure. Any single mechanism or all of these mechanisms may lead to hearing loss. Any or all of these factors together may exert an adverse effect on the inner ear without causing permanent hearing loss. The rapid recovery of normal hearing within a few days, however, seems to stand out conspicuously in importance. Labyrinthine hydrops in the course of Ménière's disease is the only pathological entity which is microscopically well established and is characterized by rapid recovery from severe nerve deafness. Recruitment is present in salicylate poisoning and in Ménière's disease. The similarity of the clinical picture of these two disorders is quite in harmony with the microscopic findings of Falbe-Hansen's experimental studies. In both conditions the increased pressure of intralabyrinthine fluids is an important feature. Therefore, in salicylate poisoning increased labyrinthine pressure, rather than direct poisoning, is responsible for the hearing loss.

The majority of available information points to interference with the normal function of Corti's organ as the immediate cause for recruitment. Increased intralabyrinthine pressure may reach a degree, which would stretch both windows to the maximum, thus reducing movement of the basilar membrane. The external hair cells, which lie directly on the basilar membrane, and which are chiefly responsible for reception of sounds of relatively low intensity, thus would be inactive due to the lack of stimulus. Sounds of very high intensity, could still penetrate through the immobilized fluid system and

reach the inner hair cells, producing recruitment. Histological changes in Corti's organ, therefore, do not necessarily take place in order to produce what we usually call nerve deafness.

The symmetrical, bilateral, identical hearing loss in this case of extreme salicylate poisoning is another factor which points to a disorder of the labyrinthine fluids, rather than to interference with the blood supply of the labyrinth. It is very hard to visualize the occurrence of such identical lesions in the intricate blood vessels of the inner ear.

Normal vestibular response in the presence of severe labyrinthine deafness is not a contradiction. The stimuli necessary for the normal function of the vestibular end organs are produced by change of position of the vestibular endolymph. For sound perception, however, vibrations numbering into the thousands have to reach Corti's organ from outside. It is not necessary to turn to another explanation frequently used, namely, that the cochlear nerve is philogenetically younger than the vestibular nerve, and therefore more vulnerable to toxic lesions.

In recent years much has been said about the close causative relationship between the use of salicylates and post-tonsillectomy hemorrhages. In this case, almost a laboratory experiment, neither hemorrhage took place nor was the prothrombin time abnormal in spite of the tremendous amount of aspirin taken by the patient. This case clearly points to two facts: 1) If aspirin plays a role in postoperative hemorrhages, it represents only one of many factors; 2) the capacity of the human body to recover from impaired blood clotting function as caused by salicylate poisoning, is quite remarkable. We should still keep an open mind on this problem as has been suggested by several workers.

SUMMARY

1. There is a strong similarity between the clinical picture of hearing loss caused by salicylate poisoning and that of Ménière's disease.

2. Falbe-Hansen's experimental studies showed increased pressure of intralabyrinthine fluids in salicylate poisoning. Increased intralabyrinthine pressure is the major histopathological feature of Ménière's disease.

3. Recruitment in both conditions is explained by immobilization of both windows caused by the increased pressure of the labyrinthine fluids.

4. An unusual case of salicylate poisoning is presented, the clinical features of which well fit into the above exposed pattern of pathology: A 28 year old patient took two hundred tablets of aspirin in six days. This resulted in severe, bilateral, symmetrical nerve deafness and recruitment. Vestibular responses remained normal. Hearing returned to normal seven days after discontinuation of salicylate medication.

180 Ft. Washington Ave.

REFERENCES

1. Covell, W. P.: A Cytologic Study of the Effects of Drugs on the Cochlea, Arch. Otolaryng. 23:633, 1936.

2. Lurie, M. H.: Animal Experimentation on Hearing, Tr. Am. Ac. Ophthal. Otolaryng. 40:375, 1935.

3. Falbe-Hansen, J.: Clinical and Experimental Histological Studies on Effect of Salicylates and Quinine on the Ear, Acta Otolaryng. Suppl. 44, 1941.

# Society Proceedings

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

*Meeting of Monday, March 1, 1954*

THE PRESIDENT, DR. ROLAND RUSSELL, IN THE CHAIR

### Otolaryngologic and Rhinologic Problems of Today

GORDON F. HARKNESS, M.D. (By Invitation)

(Abstract)

From a perusal of the literature of the past six years the essayist concludes that a problem does exist in this field. The opinions of the various authors appear to contradict the text of their own discourses. Even in teaching institutions there is acknowledgement of the problem. There seems to be lack of appreciation that the greater part of the service rendered by the otorhinolaryngologist is given in smaller communities and not in the teaching centers. There is assumption by some that the so-called "borderline" area belong to this specialty by right of eminent domain. There is acknowledgement of invasion of this field by lay organizations and professional groups lacking the degrees of doctor of medicine. There is failure to properly classify special fields within the field of otolaryngology There seems to be disregard of the fact that for years surgeons outside of the field of otolaryngology have been working in these areas.

As a partial solution of many of these factors, the essayist suggests that during his training in this specialty the otolaryngologist should receive training in some of the subdivisions of otolaryngology and the borderline areas. This would require co-operation between the various departments in teaching institutions. The young man charting his future course and the man in active practice should realize that he must stand forth not just as an otolaryngologist, but also as a maxillofacial surgeon, a plastic surgeon, a head and neck surgeon and an allergist.

DISCUSSION

DR. O. E. VAN ALYEA: I am indeed happy to laud Dr. Harkness' fine summation of the various opinions voiced throughout the country during the past few years concerning the present status and the future of otolaryngology. I think we can sense an undercurrent of optimism which is indeed gratifying. He points out the methods to obtain success and to offset some of the adverse criticisms which have been expressed. Dr. Harkness is a rare individual who has practiced in a small community without the aid of medical schools; he has attained eminence in his field by pulling himself up by his bootstraps, one might say. He has shown that a medical school at your elbow is not necessary in acquiring eminence in this field. He has outlined a schedule for the young man settling in a smaller community who has planned more or less what he hopes to attain. He does not feel that an otolaryngologist must become outstanding in all the fields that borderline the specialty, which I think is an excellent statement. I am sure we have all benefitted greatly from hearing this dissertation.

DR. ROBERT LEWY: At a recent discussion in Detroit on this subject, the point was well put by one of the speakers, who said that otolayngology is not a dying specialty, it is a changing specialty. It certainly must be true from what we hear that, at least in the smaller communities (and certainly judging by how busy people are in the larger communities) there is plenty for the otolaryngologist to do in the fields of plastic surgery, neck dissection and in allergy. It requires a broader, longer and more complex education to practice otolaryngology today than was the case twenty years ago. The question has been raised, and I think it very germaine to the problem—"Is it necessary in the average outlying community, the smaller urban centers where there are no medical schools, for an otolaryngologist to practice ophthalmology also?" This is a very practical consideration on which I should like to hear Dr. Harkness' opinion.

DR. GORDON F. HARKNESS (closing): Otolaryngology, to the lay mind and to many of the medical profession, does not include the borderline fields. To counteract that, rather than take the attitude that "I am an otolaryngologist and this is my province" we must recognize that other men of honest intentions have been working in these fields too. But we do have another approach, by overcoming that lack of knowledge of our field. We have every right to approach it, but we must not limit ourselves entirely by title to otolaryngology; we must announce ourselves as maxillofacial surgeons, or the title of whichever field we decide to enter.

As Dr. Lewy said, it is a changing specialty. As to the necessity of practicing both ophthalmology and otolaryngology in the smaller communities, there is no hard and fast rule. Communities vary. When I started in practice most of the men who were my contemporaries in Chicago also did ophthalmology and otolaryngology. It is a peculiar fact that the public will accept major surgery in otolaryngology from the young man, but not major surgery in ophthalmology. An ophthalmologist of advancing years holds his practice better than does the otolaryngologist. I do not think one can make any general statement as to whether or not it is necessary to practice both specialties, but I take the position that there is still a place in this country for the combined practice, depending on the local conditions. He is not going to be a master in either field, he may be more or less a general practitioner, but he can give good service to his community in both fields. So far as intricate and major procedures are concerned, a man's conscience must direct him as to the right thing to do for his patient, and that is what counts in the long run.

## Recording of Vestibular Nystagmus

NICHOLAS TOROK, M.D. (By Invitation)

(Abstract)

One of the major problems in vestibular physiology and pathology is the difficult clinical accessibility of the function of the end-organ. The various manifestations of vestibular excitability are largely unsuited for functional evaluation or even for simple measurement. Nystagmus, the most reliable objective expression of the vestibular excitation, has been almost exclusively considered the yardstick in the evaluation of vestibular function. Information received, however, by the observation of this phenomenon has been far from satisfactory. Especially, the duration of the evoked or reactive nystagmus cannot be expected to be the only criterion of function on which we depend for our judgment. Thus, efforts were made in recent years to extend the field of observation beyond that of the duration of the postcaloric or postrotatory nystagmus:

1. The conception of directional preponderance of the reactive nystagmus was introduced (Cawthorne-Fitzgerald-Hallpike).

2. Cupulometry is attempting to open new avenues toward a better understanding of vestibular function (van Egmond-Groen-Jongkees).

3. The study of the behavior of frequency of the postcaloric nystagmus revealed a characteristic "culmination effect" (Torok).

4. The "relation testing" takes into consideration as many circumstantial phenomena of the reactive vestibular nystagmus as possible (Kobrak).

Nystagmus recording permits a more comprehensive evaluation of the phenomenon. Simultaneous observations of various characteristics are possible. The observer may read not only the exact data concerning the duration of the reactive nystagmus, but the important properties of the frequency, the culmination reaction, anomalies of the amplitude and other minor details within one single nystagmus-component.

As early as 75 years ago reliable recordings were made in animal experimentation. A series of various attempts followed and by the use of photographic and cinematographic methods recordings were obtainable in human subjects. The discovery of the corneoretinal electrical potential was a great improvement and nystagmus recordings came much closer to the routine clinical application.

An apparatus was developed and constructed in our department based upon a working principle called "photoelectric nystagmography." This apparatus is simple to operate, requires no electrodes, causes no discomfort to the patient, and gives a record on a direct pen-writer of every detail of the eye movements.

The film presented shows the mechanism and working principle of the Torok-Guillemin-Barnothy photoelectric nystagmograph, and demonstrates the technique of recording. A short selection of graphs is shown and the evaluation of postcaloric nystagmus is discussed.

### DISCUSSION

DR. SHERMAN SHAPIRO: Many years ago I became interested in nystagmography and combed the literature on the subject pretty well because I dreamed of getting something tangible that could be put on a graph. I did not find anything that could be used in a practical way, although a tremendous amount of work had been done in this field, particularly by Ohm in the German literature; he spent a lifetime working with little mirrors on the sclera, and so on. Tonight's demonstration is not yet a clinical method, but is a remarkable piece of work from a research standpoint, one of the best that has

come from this locality. It holds a great deal of promise, and I think further investigation should be subsidized and carried through.

There is one difficulty about all these studies which makes them uncertain to work with, and that is, of course, the voluntary movements. The patient will move his eye, and that must be integrated into the chart. I think Dr. Torok should be congratulated. He has been working on this for years and should be greatly commended on what he has achieved so far.

Dr. Alfred Lewy: The record of the type and duration of the nystagmus, as well as the difference in the quality of the nystagmus, is very interesting in the diagnosis. This may perhaps help a great deal more than we realize today in the diagnosis of intracranial space-occupying lesions. In the meantime, a very good practical technique is one which I have advocated for many years. It does not have the disadvantage that the minimal method has, that is, if it does not work it must be tried again with a little more ice water, and so on. That is appreciated by the patient. I use 50 cc of water at 60° temperature, and I have never seen a case in which nystagmus could not be produced if it could be done by any other variation of the caloric method. On the other hand, I have never known it to produce nystagmus out of the average normal, which is about 60 to 120 seconds, that would be shown by any other caloric method. From a scientific standpoint Dr. Torok's presentation is a great advance, it looks very practical, and I hope it may help in the diagnosis of intracranial injuries and tumors. The other vestibular tests, such as postural nystagmus, are of great importance in the diagnosis of head injuries, showing whether or not more than the skull has been injured.

Dr. Nicholas Torok (closing): I wish to thank you for your comments. In reply to Dr. Shapiro, voluntary eye movements, particularly blinking, are occasionally disturbing in the recording of the nystagmus. Before calorization is performed a quick adjustment must be done which provides ample opportunity to the patient to become acquainted with the procedure. In exceptional cases a second recording may be necessary to avoid disturbing factors in the record.

In reply to Dr. Lewy, not only 50 cc of 60° water but one or two cubic centimeters of water a few degrees cooler or warmer than the body temperature may produce sufficient stimulation to cause vestibular nystagmus. The newest methods of minimal stimulation by rotation (cupulometry) have revealed similarly that a velocity of two to three degrees per second produces measurable vestibular stimulation.

## "Filmettes" As A Teaching Aid In Otolaryngology: Motion Picture

MARVIN J. TAMARI, M.D. (By Invitation)

AND

RICHARD A. BUCKINGHAM, M.D.

(Abstract)

This demonstration movie shows how short lengths of motion picture film, called "Filmettes," depicting diseases of the ear, nose and throat, are used for undergraduate and postgraduate teaching at the University of Illinois and the Illinois Eye and Ear Infirmary.

More than 40,000 outpatient clinic visits are made at these two institutions each year, and many rare and unusual conditions are seen. As many of these interesting cases as possible are photographed. The films are kept in the files until needed for a lecture or for presentation at one of the weekly departmental seminars. With 25, 50 or more feet of motion picture film taken of typical and atypical ear nose and throat conditions, the clinical presentations are more realistic than they would otherwise be. Little editing is done on the films, although an occasional rare condition is titled to preserve it for future presentation. Thus expense is minimized and the maximum benefit is derived from the film.

Several of these filmettes have been spliced together and titled for presentation at this time. They are as follows:

1. Hemotympanum: this occurred following a postnasal pack for severe epistaxis.

2. Acute otitis media before and after myringotomy. A view of the drum ten days later shows no evidence of the site of the incision. The ear drum pictures were taken with the Holinger-Brubaker camera through the courtesy of Dr. Paul H. Holinger.

3. Positive fistula sign in case of chronic otitis media with cholesteatoma. The patient demonstrates past-pointing and nystagmus. A labyrinthine fistula was found at surgery.

4. Cerebrospinal rhinorrhea due to tumor of the middle cranial fossa with destruction of the sella turcica. A simple means of establishing the presence of cerebrospinal fluid is demonstrated. One cubic centimeter of the secretion is collected in a test tube and a clinitest tablet added. Since normal cerebrospinal fluid contains 50 milligrams per cent glucose, a greenish color appears in the test tube.

5. Maggots in the external canal, probably Wohlfahrtia species. The patient had a neglected chronic otitis media which served actually as a breeding place for these maggots.

6. Carcinoma of the lateral margin of the tongue. This shows a typical carcinomatous lesion. In addition the mobility of the tongue is demonstrated. This case was operated on soon after photography. A hemiglossectomy, hemimandibilectomy, and radical neck operation were performed.

7. Facial nerve paralysis following mastoid surgery unsuccessfully treated with a masseter muscle transplant. This illustrates inherent faults in the procedure. Such a photographic record should discourage any attempts to revive the operation.

8. Bleeding of the lateral sinus encountered during mastoidectomy. While an ordinary mastoidectomy for chronic middle ear disease was being photographed, the bleeding occurred. This being a rare occurrence during photography, it is included.

9. Chronic otitis media with nystagmus and lateral gaze to affected side. A cerebellar abscess was suspected and a radical operation performed within an hour after the photograph was taken. The cerebellum was aspirated through the dura of the posterior fossa and pus was obtained. A drain was left in place in the abscess.

THE PRESIDENT, DR. ROLAND RUSSELL, IN THE CHAIR

*Meeting of Monday, April 5, 1954*

## Morphologic Changes In the Mastoid Bone Under Antibiotic Therapy

MARVIN J. TAMARI, M.D. (By Invitation)

AND

PAUL B. SZANTO, M.D.

(Abstract)

It is well known that the clinical course of otitis media is changed by the use of various antibiotics. However, correlation of the altered clinical course with the histopathologic changes has not received wide attention. "Masking"—that phase of latency in regard to signs and symptoms which is observed prior to either resolution or complica-

tion—often characterizes otitis media under antibiotic treatment. In investigating the histopathologic picture present during the period of latency, two sources of information have been examined. Those cases which, in spite of antibiotic therapy, came to surgery, constituted a direct approach. For comparison sections of not operated mastoids and sections of bone material not influenced by antibiotics have been studied.

Masking of symptoms or latency in otitis media can occur in the course of otitis in infants, in serous otitis media, tuberculosis, in acute otitis media caused by pneumococcus type III (streptococcus mucosus) and in otitis media treated with antibiotics. The histopathologic changes are characterized by the presence of abundant fibrous exudate in the pneumatic cell lumina and in the adjacent marrow tissue spaces. It consists mainly of fibroblasts, histiocytes, plasma cells and collagen, in contrast to the polymorphonuclear leukocytic accumulation in the trivial form of otitis media.

Pneumococcus Type III otitis and otitis treated with antibiotics exhibit identical changes. In both instances abundant fibrosis prevails. Production and condensation of collagen is in the foreground. The pneumatic cells become constricted by formation of new bone at the walls of the cell and composition of rootstock bone in the center of the cell. The period of bone rarefaction appears to be shortened or absent. In Pneumococcus Type III infection, the latency becomes interrupted with disappearance of the "slime layer" of the capsulated coccus; in otitis treated with antibiotics it becomes manifest through a superinfection or reactivation of resistant strains and as yet unknown enzymatic action.

In the past five years three types of clinical course of treated otitis media have been observed:

1. Where adequate antibiotic has been given early in the disease and drainage has been established either spontaneously or by paracentesis, the acute otitis subsides after four or five days with return of normal function.

2. There may be a protracted course when the administration of antibiotics is insufficient in dosage or duration. There may be intermittent discharge and the otitis may subside leaving a marked, permanent hearing loss.

3. Where early antibiotic treatment controls symptoms and is discontinued early the otitis may pass into a state of latency similar to that of the pneumococcus Type III infection. These cases usually had a surgical outcome, either because of extensive destruction or complication.

Those cases which fall in Groups 1 and 3 have a favorable outcome—spontaneous or surgical resolution. We cannot predict the outcome of those cases which have a protracted course with extensive scarring following poorly administered antibiotics. However, if we compare the ages and the type of surgery being done in the antibiotics era as compared with the previous era, we find that now more patients in younger age groups are undergoing surgical care for chronic changes than in the past. The series, however, is not sufficient to draw any general conclusions at this time.

DISCUSSION

Dr. Thomas C. Galloway: Dr. Tamari is to be congratulated on this beautiful study. It would be fine if we could determine the last point of reversibility of the pathologic process and how completely we could rely on antibiotics for a cure. Fifteen years ago I gathered tissue blocks from mastoids to attempt to determine this. The practical difficulties were so great that the work was not completed. Dr. Tamari has given us a basis for an answer, but there are several factors that complicate the question, especially the developing resistance of invading organisms.

A case in point was a patient operated upon three weeks ago and now home. After one week of otitis media she developed meningitis with crossed spasticity and hemilateral sixth and seventh nerve involvement and coma. Under antibiotic therapy the symptoms subsided except for slight ear discharge. At operation on a sclerotic mastoid I could not decide whether she might have recovered without intervention. Dr. Davis, our pathologist, believed she would not because of osteitis and multiple small abscesses. From past experience we know that the danger of recurrence in such patients is so great that we should not risk leaving an infected focus. Before we knew much about petrosal infection, I saw, with Dr. Lawson an otogenic meningitis. That patient was relieved by intracarotid Pregl's iodine of all findings of meningitis due to hemolytic streptococcus. There was recurrence of meningitis after three weeks and, at autopsy, a small focus of osteomyelitis was found at the apex of the petrosa.

Dr. Pierce Theobald: This paper brings home the fact that antibiotics are not all they seem to be in the treatment of middle ear infection. If some of you have not had experience with that fact, I am sure you will soon. I had such an experience about two months ago. A six year old boy was admitted to the hospital with acute otitis media. Paracentesis was performed and sensitivity tests were done and the appropriate antibiotic given. The drum membrane healed and there was no evidence of discharge in the middle ear, but

a post-auricular abscess developed. After incision the material obtained was cultured and sensitivity tests made to determine the proper antibiotic to use. The fistula which developed continued to drain and after two weeks or so the temperature continued to spike and there were changes seen by x-ray. At surgery the entire mastoid cortex was found to be destroyed by disease. Ordinarily I use an electric drill, but there was no need to do so in this case. A curette could be introduced easily and, on approaching the lateral sinus, it also was destroyed, as was the tegmentum tympani. The patient was threatened with intracranial complications but he had an uneventful recovery after surgery.

Dr. Robert Henner: I should like to congratulate Dr. Tamari on the excellent correlation between the clinical picture and the pathologic changes that take place in treatment of mastoiditis with antibiotics. The cases he illustrated came from those in which antibiotic therapy failed and for that reason any generalization would be difficut. We find in treatment of otitis media, a group who are wholly dependent on antibiotics and we feel completely secure with their use. I think this treatment is to be criticized when adequate dosages are not given, and particularly when the temperature drops and no further attention is given to the case. These cases may come to attention later through hearing surveys or secondary complications, but if the pathology of fibrosis is common they may be seen later with a permanent loss of hearing. Also, as Dr. Theobald pointed out, there are the rare cases which continue to have necrosis of bone, and complications. The principles which Dr. Tamari's pathologic report should emphasize are the continuing of antibiotic therapy until hearing returns and resolution is complete. Only then can one feel safe in stopping the treatment.

## Changing Concepts In Rhinoplastic Surgery

Maurice Cottle. M.D., (By Invitation)

(Abstract)

The essayist illustrated and elaborated upon the following points:

1. Combining pyramid and septal surgery must be a procedure accepted, approved and easily performed by the rhinologist.

2. Shortening the nose is not only the shortening of the tip length, according to Joseph, or by rotation of the lobule according

to Fomon et al, but also a diminution of the nasal height changing the nasal index.

3. Cutting the upper lateral cartilages to aid shortening of the nose (according to Joseph) cannot be replaced by doing little or nothing to them, but must be replaced by a reconstruction based on exact structure and function information.

4. Inspection and photography of the base of the nose with and without retracting instruments and with the head in several planes help in diagnosis of functional deficiencies.

5. Study of nasal valves in vivo, at operation, and on cadavers, elucidates tension-collapse of the upper laterals, ballooning of the upper laterals and their relationship to sleep and rest.

6. In connection with the above the concept of using the upper laterals to correct a small saddling of the cartilaginous vault becomes unacceptable.

7. The conception of the removal and disposal of a hump from a nose is replaced by one which offers restoration or repair of the nasal roof.

8. Narrowing a nose is now part of a conception of changing the nasal aperture, to be distinguished from the pyriform aperture.

9. The floor of the pyriform aperture has a varied structure but is always characteristic of the ethnic origin of the individual. The vestibules of the lobule must conform to the patterns dictated by the underlying bone.

10. The external nasal pyramid is an attic with a considerable air space, quite empty, covered by an insulating roof. It offers protection to the internal nose against outside influences and helps retain the moisture and warmth of expired air for the benefit of the nasal mucosa.

11. Therefore, any deformity, injury or disease of the external nasal pyramid making rhinoplasty or rhinoplastic procedures necessary, becomes the responsibility of the rhinologist who is dedicated to prevention and treatment of nasal diseases.

# Abstracts of Current Articles

## EAR

**Auditory Training of Severely Hard of Hearing Pre-School Children—Results from a Swedish Series Comprising 36 Children.**

*Wedenberg, E.: Acta Oto-laryng.*, Suppl. 110.

The objective of auditory training is to provide a sufficient command of speech which in verbal testing demonstrate normal intelligence. Children were classified in five groups:

1a. High frequency loss with retained hearing for low frequencies.

1b. High frequency loss with poor hearing for low frequencies.

2a. Flat hearing loss.

2b. Excessive flat hearing loss.

3. Vibration sense in low frequencies, rising hearing for high frequencies.

Hearing aids are not used until the child has acquired a small vocabulary through speech "ad concham." Speech reading is not introduced until the child has acquired the "listening attitude."

The author's method emphasizes the auditory sense, with the visual sense as a complement, adapting the hearing of speech to a decoding of sound features of minor importance in the normal hearing person. This is accompanied by use of spectral composition of speech sounds. This should result in interweaving of auditory and visual perception so "that the child is unable to say 'I hear' or 'I see' but rather 'I perceive'."

HILL.

**A Method to Test the Intelligibility of the Speech of Pre-School Children with Severe Hearing Impairment.**

*Bjuggren, G.: Acta Oto-laryng.*, Suppl. 110.

This is a preliminary report of a method aimed at eliminating as prerequisites, reading ability and behaviour stability in subjects tested. The picture-book technique, using two auditors, and in some

cases tape recording, was employed. Intelligibility increases in proportion to the acquired vocabulary, as shown in longer sentences, resulting from auditory training.

HILL.

**Speech Audiometry—An Experimental and Clinical Study with Swedish Language Material.**

*Lidén, G.: Acta Oto-laryng.*, Suppl. 114.

This investigation was carried out with the objective of developing a speech audiometer for clinical use and standardizing test material in the Swedish language. While hearing acuity at the hearing threshold can be fairly well determined from pure tone audiometry, ordinary speech is perceived at a higher level. Consequently "only speech audiometry can demonstrate satisfactorily what difficulties are present there for the hard-of-hearing person."

In this monograph of some 138 pages the author has covered the subject of speech audiometry in a most meticulous manner. Considerable space is allocated to clinical investigations, such as the value of discrimination tests in otosclerosis, Ménière's disease, etc. He concludes that speech audiometry is equally as important as pure tone audiometry and that both methods together determine the patient's degree of handicap.

It is interesting to note that acoustic trauma is assuming increasing importance as a basis for compensation in Sweden just as it is in this country.

HILL.

**The Mechanism of the Fistula Test.**

*Dohlman, G.: Acta Oto-laryng.*, Suppl. 19, pp. 22-26.

It is generally accepted following Zwergius' work that the fistula test is a reaction of ampullar crista of the semi-circular canals, that movement of the endolymph is necessary and that free movement of either the oval or round window was essential to a positive reaction. However, "if the membranous labyrinth is to be regarded as a closed membranous sac in the perilymphatic spaces the only effect of an altered pressure at the site of the fistula will be a raised or lowered hydrostatic pressure but no endolymph movements." Furthermore Zwergius found a difference in the response to the test according as to where the fistula was located. When near the ampulla nystagmus

in the plane of the canal was elicited on pressure but when the fistula was at the nonampullated end of the horizontal canal, pressure elicited nystagmus in the plane of the anterior vertical canal.

The author's studies suggest an entirely different conception of the hydrodynamics involved. Rather than increased pressure at a fistula near the ampullated end of the canal causing free perilymph flow to the region of the windows, any movement meets obstruction from the ampullary vault. Likewise, flow towards the non-ampullated end is blocked by the widened endolymphatic canal near its opening into the utricle. He suggest that inflammatory changes in the interior of the semicircular canals may result in obstruction to movement of the perilymph, causing endolymph displacement and cupular deflection due to pressure changes at the fistula such as to produce either the regular or reversed reaction.

HILL.

**Acute Cryptogenic Deafness Treated by Fenestration.**

*Nielsen, J. C.: Acta Oto-laryng.*, Suppl. 109, pp. 122-124.

Acute cryptogenic deafness has been attributed to neuritis of the eighth nerve, injury of the central nervous system, hemorrhage, thrombosis, and embolism. Mygind advanced the hypothesis that this was due to acute pressure in the labyrinth and that fenestration might restore hearing. The author adds two case reports to the three previously reported by Opheim: one, operated on the 6th day, had relatively good restoration in the lower frequencies; the other, who underwent operation on the 11th day, regained only slight hearing confined to the low frequencies.

The author postulates that operation must be performed early if complete deafness is to be avoided.

HILL.

**The Cochlear Microphonic Potentials During Sympathetic Stimulation.**

*Krejci, F. and Bornschein, H.: Acta Oto-laryng.* 44:2:154-156 (Mar.-Apr.) 1954.

Experiments on cats subjected to prolonged stimulation of the cervical sympathetic trunk and also the stellate ganglion fail to cause a decrease in the cochlear potentials. Local anemia must be extremely severe to cause diminution of potentials—more than seems possible from vaso-constrictor stimulation.

HILL.

**Hearing Impairment Due to Electrical Insult.**

*Neuberger, F.: Monatsschrift fuer Ohrenheilkunde und Laryngo-Rhinologie, Wien* 87:187 (Apr.-June) 1953.

Cases of hearing impairment are rare among the few survivors of high voltage accidents, in the author's experience. He describes the clinical picture and the possible pathology of a 35 year old man, who survived a 20,000 volt shock in a railroad workshop.

Immediately following the momentary gripping sensation the patient experienced marked tinnitus and hearing loss. He never lost consciousness. Both tinnitus (roaring and ringing) and hearing loss increased gradually and symmetrically in both ears, as given by subjective description and as tested by repeated audiograms over the following period of time. The hearing loss was described as retro-labyrinthine or nerve type. After one year of intensive treatment with vitamin B and vasodilators it had shown no improvement.

Three possible pathological theories are offered:

1. Direct damage of the ganglia due to vasomotor disturbances caused by the strong current.

2. Serous meningitis as a result of the meningeal hyperemia and edema following the electrical insult, causing damage of the acoustic ganglion *after a latent period.*

3. Theories 1 and 2, simultaneously.

In the opinion of the author, the described case represents Theory 1 (direct damage of the ganglion in the medulla), since tinnitus and deafness began immediately, rather than following a latent period as noted in the meningeal Type 2.

FISCHER.

**Congenital Deafness.**

*Van Egmond, A. A. J.: Journ. Laryngol. and Otol.* 68:429 (July) 1954.

The subject material is classified as:

1. Congenital e.g. from rubella during pregnancy.
2. Hereditary e.g. otosclerosis.
3. Congenital and hereditary e.g. sporadic recessive deafness.

Congenital hereditary deafness is further classified as:

A. Dominant inner ear deafness which occurs in middle age, progresses rapidly and affects most frequencies. This appears in each generation, and may involve 50 per cent of the offspring.

B. Congenital sporadic recessive deafness which though sporadic in occurrence is much more common in consanguineous marriages and accounts for seven per cent of all deaf mute children.

The genetic and anatomical aspects of these two conditions are discussed and it is suggested that marriages involving one congenitally deaf person should be undertaken only after careful investigation of both person's family trees. Marriage between two congenitally deaf persons should be discouraged; and even more so, consanguinity.

Additional causes of congenital deafness including atresia auris congenita, erythroblastosis fetalis, maternal rubella, syphillis, cretenism, and drugs among others, are discussed.

SOOY.

**Hydrocephalic Disturbances Following Mastoidectomy.**

*Bauer, E.: Abstract Monatsschrift f. Ohrenheilk.* 87:263 (Oct-Dec.) 1953.

Meningeal irritations or inflammations usually first cause increased cell count and later increased pressure of the cererospinal fluid. The normally somewhat different permeability of the cuboidal epithelium of the choroid plexus in children may in case of such meningeal irritation first cause increased production of spinal fluid, and second, the appearance of cells. Many times, this fact remains unnoticed, as normal C.S.F. drainage and normal absorption by the Pacchioni granulations compensate for this increase in production. Assuming normal intrathecal flow, increased production of C.S.F. must lead to hydrocephalus, however, if the above named drainage and absorption is inhibited.

Two examples of such abnormal conditions are presented. Both patients are children, who underwent a simple mastoidectomy because of acute otitis media complicated by acute mastoiditis.

In Case 1 an extradural abscess was found. During the postoperative course, the continued elevation of the temperature led to the detection of an elevated pressure of the otherwise nonpathological C.S.F. as the causative factor. The patient had been treated intensively with antibiotics. The condition cleared up spontaneously

after two weeks, and three lumbar punctures were done to relieve the pressure and thus bring the temperature down.

Traction on the meninges caused by the extradural abscess was given as a possible explanation. This traction, it is stated, was not immediately relieved by the evacuation of the abscess. Normal conditions returned only gradually, and the spinal fluid drainage remained inhibited for some two weeks in spite of intensive antibiotic therapy.

In Case 2 a peri-sinus abscess causing sinus thrombosis was found in addition to a large extradural abscess. The latter was evacuated and the thrombus was removed. The cavity was packed tightly. Continued elevation of the temperature and vomiting in spite of antibiotic medication gave indication for a lumbar puncture. The C.S.F. pressure was found to be enormously high. Cell count and serology were normal. The condition returned to normal spontaneously after four days.

The author's explanation follows: Two causative factors were apparent in Case 2. The extradural abscess may have caused traction as in Case 1. In addition, the tight packing of the sinus caused impairment of the venous drainage of the brain. This latter factor is mentioned as particularly possible in cases of an abnormally narrowed jugular foramen of the other side (left in this case). The author does not state whether this possibility was investigated by roentgen films of the cranial foramina.

FISCHER.

**Efferent Nerve Fibers of Cochlea.**

*Portmann, Michel and Portmann, Claudine*: A. M. A. *Arch. Otolaryng.* 59:543-554 (May) 1954.

The authors describe a set of fine, distinct nerve fibers originating in the medulla and terminating at the inner hair cells, which they conclude are efferent nerve fibers. The fibers arise in a nucleus composed of small cells located in the region of the superior olivary complex, near the dorsal edge of the accessory olivary nucleus and the trapezoid body, and they are directed dorsally toward the deep facial knee of the same side, all of them crossing the median line and emerging from the medulla between the vestibular nerve and the nervus intermedius, and then continuing between the two portions of the vestibular nerve. After rejoining the cochlear nerve, they coil in a

spiral around the ganglion of Corti up to the helicotrema, terminating at the various turns, leveling with the internal spiral plexus.

The Portmanns affirm that the inner hair cells have a double innervation: an afferent innervation, formed of radical fibers, and an efferent innervation, composed of spiral fibers. The outer hair cells are almost exclusively innervated by the well-known afferent fibers.

The writers substantiate the fact that the organ of Corti is innervated by a double system of nerve fibers: the sensory fibers, already known, and the efferent fibers, the latter constituting a bundle clearly described in its bulbar portion as early as 1942, by Rasmussen. Weber's technique of fixation and silver impregnation allowed the authors to determine with precision the peripheral topography of the fibers and their terminations at the level of the inner hair cells of the organ of Corti. Thus they are able to bring together into one general plan heretofore isolated histologic observations.

The efferent nerve fibers do not present any relay along their course. Their topography, morphology and behavior under acoustic traumatism attest to their efferent nature, however. On the author's sections, the spiral (efferent) fibers were always lighter in color than the sensory fibers, in any part observed, and in any species (rat, cat or guinea pig) studied, the spiral fiber remained unchanged but sensory fibers degenerated when subjected to acoustic trauma.

These facts are offered as formal proof of the efferent nature of the fibers but their function has yet to be established.

HILDING.

**Tumors of the Jugular Glomus.**

*Alcázar, L. M. V. and Vila, P. A.: Acta Otorinolaryngol. Ibero-Amer.* 5:4:294-305, 1954.

Tumor of the jugular glomus or "non-chromaffin paraganglioma" of the middle ears, affects the temporal bone, usually the adventitia of the cupula and jugular foramen, immediately below the floor of the middle ear and near the tympanic branch of the glossopharyngeal nerve. However, other locations may be involved. The glomus is usually a single ovoid or flattened body 0.5 mm long and 0.25 mm wide, however, it may consist of two or three small bodies. It is composed of capillary blood vessels separated by numerous epithelioid cells. It is innervated by the glossopharyngeal nerve. The blood supply is from the ascending pharyngeal artery. Its function is the control of local circulation.

Tumor of the glomus may be caused by trauma or by endocrine disturbances, although its etiology is not established. It usually occurs in adults and is generally unilateral. In 80 per cent of reported cases it has occurred in women. Macroscopically the tumor appears to be benign and noninfiltrating. It develops slowly but progressively, causing erosion of the surrounding bone and eventually effecting the middle ear, invading the tympanic membrane, the external auditory canal, and later extending to the facial nerve and labyrinth. Microscopically the histologic structure resembles that of tumors of the carotid body. No atypical mitosis is noted suggesting malignancy. It is characterized by chains of epithelial cells with an alveolar structure. The cells are usually large and polyhedral, with either a small single nucleus or two or three nucleoli. It contains no chromaffin substance. It is profusely innervated and is frequently mistaken for hemangioendothelioma.

Symptoms develop slowly and consist at first of conduction deafness and pulsating buzzing noises, which may be audible by means of a stethoscope applied to the external ear. There is a sensation of obstruction in the ear similar to that of obstruction of the tubes. There is frequently a discharge due to secondary infection, resembling that in chronic suppurative otitis. When the tumor invades the labyrinth, deafness of perceptive type occurs. Eventually facial paralysis follows. Vertigo and nystagmus may be noted, but otalgia and headache are rare, except when cranial hypertension supervenes, in the terminal stage.

Otoscopy reveals reddening of the posterior lower portion of the tympanum. In some cases a large polyp forms in the floor of the passage and protrudes from the auditory meatus. It is a pulsating, round, smooth reddish mass which bleeds readily.

Tumors within the ear result in erosion of the posterior condyle, manifested by difficulty in swallowing, hemiplegia of the velum and palate, and larynx, paralysis of the trapezius and sternocleidmastoid muscles and deviation of the tongue. Occasionally a cervical swelling, with fremitus, may be noted below the ear.

The roentgenographic picture is that of calcium absorption from the bone and erosion of the floor or inner wall of the cavity. Arteriography reveals the profusely vascularized tumor in the posterior fossa.

The condition is inevitably progressive, but metastases are rare. The absence of pain is a diagnostic factor which excludes cancer of the middle ear. Biopsy differentiates the tumor from angioma and angiography will exclude aneurysm of the internal carotid. Occa-

sionally the symptoms of endocranial invasion may be mistaken for those of cerebral abscess.

External tumors require surgical extirpation; internal types respond to radiotherapy. Sclerosing treatment and cauterization with silver nitrate are ineffective and treatment with androgens is of questionable value. In view of the slow development of the tumor, it can safely be kept under observation for some time. However, when it begins to increase in size immediate and complete extirpation is essential. Electrocoagulation may take the place of surgery in some cases. Blood transfusions are frequently necessary. Ligation of the carotid prior to operation has not yielded the desired results. Opinions differ as to the radiosensitivity of the tumor. X-rays may be helpful in checking recurrences, following surgery, or prior to intervention, in order to decrease the vascularization of the tumor. Ligation of the external carotid and jugular may check recurrences, by shutting off the blood supply to the tumor. This decreases the symptoms, especially the pulsating buzzing noises.

The prognosis is unfavorable, especially in the internal type, because of the tissue destruction and the tendency to recurrence. The facial paralysis is usually permanent. However, if the condition is recognized early and adequately treated, recovery may be complete. A case is cited in which the application of 5,000 r resulted in disappearance of the symptoms.

HIGBEE.

**Minor Surgery to Restore Hearing in Otosclerosis. Mobilization of the Stapes. Clinical Cases.**

*Rosen, S.: Acta Otorinolaringol. Iber-Amer.* 5:3:180-187, 1954.

Ankylosis of the stapes, which impedes the transmission of sound to the cochlea, has been considered to be permanent and irreversible. Fenestration of the labyrinth to restore hearing is usually performed.

The author suggests a new method of treating otosclerosis by which the base of the stapes impacted in the fenestra ovalis can be mobilized by means of a probe inserted through the external auditory canal to restore the normal function of the chain of ossicles, and thus to restore hearing. The articulation of the stapes and the incus is explored by means of an auricular speculum, using a Zeiss binocular lens for visualization. The technique resembles one which has been advocated for section of the corda tympani in Ménière's disease.

Three hours prior to the operation the patient is given 3 mg of nembutal, followed two hours later by 10 mg of demerol. To induce local anesthesia, 1 per cent novocaine with adrenalin is injected subcutaneously into the upper posterior wall, the anterior portion, and the roof of the external auditory canal. The skin is separated from the bone as far as the membrana tympani. The upper half is freed from its sulcus and is folded upward over it like an apron, exposing the chamber of the middle ear. In 85 per cent of cases the articulation of the stapes and incus is immediately visible. If not, it is necessary to remove 2 or 3 mm of the posterior margin of the bony canal above the incus, exposing the entire stapes, and the opening of the fallopian canal. The degree of mobility of the stapes is then determined by slight downward pressure with the probe on the apophysis of the incus near its articulation with the stapes. If the stapes is firmly impacted only the apopysis moves, and the stapes and its tendon remain fixed.

To free the impacted base of the stapes, a dental hook is inserted above the articulation. Downward pressure in the direction of the tendon results in sudden freeing of the base. When slight downward pressure is exerted on the upper surface of the incus near its articulation with the stapes, the head, neck, crura and tendon of the stapes move as a unit, and hearing is restored. The intact tympanic membrane is then replaced in its normal position. The patient can be discharged on the following day. No vertigo occurs, and no postoperative treatment is required. In most cases the method is successful in restoring hearing permanently. The ankylosis does not recur. If the mobilization should fail, fenestration may be carried out. Five cases are cited in which this method was used with excellent results.

HIGBEE.

**Neoplasms of the Middle Ear and Mastoid. Report of Fifty-Four Cases.**

*Bradley, W. H., and Maxwell, J. H. Laryngoscope* 64:7, 533-556 (July) 1954.

This report is based upon a clinical analysis of 54 patients with neoplastic disease of the middle ear and mastoid observed from 1917 to 1952. Follow-up studies in each case are complete to January 1953. An incidence of one tumor of the middle ear or mastoid to each of 7,777 patients seen in the ear, nose and throat department and one case of carcinoma on 208 cases of chronic suppurative otitis media occurred in this series.

The data presented here substantiates previous findings, in that malignant neoplasms of the middle ear and mastoid occurred more commonly in older age groups while sarcomata were seen more frequently in children. Glomus jugulare tumors were confined mainly to the age brackets 40 to 45 years and benign neoplasms occurred primarily in patients less than 30 years of age. In all groups the incidence is equally divided between the sexes except in the case of the glomus tumors where the ratio was 2:1 in favor of the females.

Although long continued chronic suppurative otitis media was found in association with carcinoma in almost one half of the cases, the authors suggest that their findings strongly indicate that other factors must be involved.

The glomus jugulare tumor is considered as a benign tumor in this series even though there are some malignant variants. Also neurofibromas of the facial nerve are included since they are essentially middle ear lesions. Osteomas of the mastoid were included but exostoses and pedunculated osteomas of the external auditory canal are omitted.

When the symptoms and signs of these tumors were examined it was found that pain out of all proportion to the apparent extent of bone disease or amount of purulent discharge is one of the more common but not necessarily early symptoms. Bleeding from apparent granulation tissue should make one suspicious of malignancy. Any aural polyp found in a suppurating ear should be removed and subjected to histopathological examination.

There were no characteristic early clinical manifestations in carcinoma of the middle ear. Facial paralysis was a common finding and may occur early. This finding, with labyrinthine irritation and perceptive deafness in a patient with chronic suppurative otitis media, is an indication of extension of the disease beyond the confines of the middle ear and the pneumatic structure of the mastoid.

Early diagnosis of benign tumors of the middle ear demands painstaking study. Various diagnostic aids are necessary such as palpation of the tympanic membrane and even the hazardous myringotomy and biopsy.

No form of therapy of the malignant neoplasm has been very successful. Radical surgical excision and irradiation have been the only therapeutic procedures of any value. The treatment used in

this series was radical surgical excision of the neoplasm whenever possible. In the majority of patients intensive x-ray therapy was administered postoperatively, while in a few highly anaplastic lesions irradiation therapy alone was used.

The treatment of the benign tumor was very successful. Eight of the glomus tumors were removed surgically and two were given x-ray therapy with uniformly good results. The neurofibromas of the facial nerve were removed and the nerve repaired with a free graft with satisfactory functional results.

SENTURIA.

## NOSE

**Nasal Carriage in Staphylococcal Skin Infections.**

*Tulloch, L. G.: B. M. J.* (Oct. 16) 1954.

The author suggests that the treatment of chronic staphylococcal infection of the skin rarely results in a permanent cure despite the use of modern antibiotics because of several reasons. One of these he believes is reinfection of the skin by staphylococci flourishing in the anterior nares.

Since it has been shown that staphylococci may be cultured from the noses of healthy individuals, the similarity of the types occurring in the nose and in the skin infections would have to be proved. Previous investigators had shown that strains from the nose and intact skin were identical in a high percentage of cases. This investigator sought to prove the identity of strains obtained from the nose and from the site of skin infections in the same individaul.

Seventy-three pairs of cultures from the noses of patients suffering from sycosis barbae, furunculosis, folliculitis and infectious eczematoid dermatitis were collected. These were phage typed. Where both strains were typable the strains were identical in 95.2 per cent of cases of sycosis barbae, 81.8 per cent furunculosis and folliculitis and 87.5 per cent of infections eczematoid dermatitis.

Although this is a small series of cases this report forms one link in the chain of evidence which directs attention to the anterior nares as a possible source of re-infection in various chronic dermatoses. In

view of these findings it may be desirable for rhinologists to give more thought to the nose as a potential source of infection and contamination of the surrounding skin and environment.

BOIES.

**Malignant Tumors in Nasal Adhesions.**

*De Arratia, F. F.: Acta Otorinolaringol. Iber-Amer.* 5:3:209-212, 1954.

Obstructive cicatricial nasal adhesions may be membranous or bony. Occasionally the deformity is congenital, but as a rule it results from surgical intervention, cauterization, prolonged retention of a foreign body in the nares, with consequent erosion of the mucosa, or from other injuries. The author stresses the need for postoperative prophylactic care to avoid the formation of adhesions which might predispose to malignant tumors.

Two cases are cited in which sarcoma developed on the site of postoperative nasal synechiae, resulting in interference with respiration, neuralgia, sneezing, tinnitus, etc. In the first case, the sarcoma invaded the maxillary sinus. The patient was 25 years old and in good physical condition. After the tumor was extirpated postoperative radiotherapy was given. Recovery was complete. When the patient was last examined, two years following surgery there had been no recurrence.

In the second case pulmonary metastases had occurred. The patient, a man 68 years of age, was in poor condition. The malignant process, a fibrosarcoma, had originated in an intranasal adhesion. It had invaded the maxillary sinus, the orbit, and the velum of the palate. Because of the metastases, no favorable results could be expected from surgery. Radiotherapy alone was employed as a palliative measure.

Early in the development of this condition there are no symptoms, except nasal obstruction and epistaxis. Later on a malodorous secretion, loss of olfactory function on the affected side and trigeminal neuralgia develop. As it progresses, the tumor may involve the orbit and the cranial cavity, may displace the nasal walls and cause a bulge in the cheek and may invade the palate and sinuses. The neoplasm can be differentiated from benign tumors by anterior and posterior rhinoscopy, roentgenography and biopsy, which should be carried out in any case in which there is the slightest suspicion of malignancy.

HIGBEE.

## LARYNX

**Rehabilitation After Laryngectomy.**

*Jesberg, N.: Calif. Med.* 80:90 (Feb.) 1954.

The author presents his experiences with speech rehabilitation in 109 laryngectomized patients and two individuals with severe laryngeal injuries.

The teaching of esophageal speech should be instituted early even on the fifth to sixth postoperative day, or as soon as the patient is able to swallow. The use of carbonated beverage is helpful. Better results are obtained in relaxed patients and when the cannula has been removed.

The most effective teaching was done by a laryngectomized patient with good esophageal speech.

Sooy.

**The Incidence of Tuberculosis in Patients Suffering from Cancer of the Larynx.**

*Niccoli Vallesi, R.: Bollettino Delle Malattie Dell' Orecchio, Della Gola, Del Naso,* 71:146 (Mar.-Apr.) 1953.

Two hundred and sixteen patients with proven squamous carcinoma of the larynx were investigated histologically for evidence of concomitant tuberculosis and none were found.

Three patients (1.38 per cent) had involuted tuberculosis involving the lungs or lymphatic glands.

Nine patients (4.16 per cent) showed inactive lesions of the lung or pleura and nine patients (4.16 per cent) had histories suggesting tuberculosis. The authors conclude:

1) There are no clinical elements suggesting an antagonism between cancer and tuberculosis.

2) The two diseases can coexist without any change in their respective courses.

3) The two conditions when they coexist do so on the basis of pure chance.

4) The scarcity of simultaneous tuberculosis and cancer of the larynx is due to the diverse frequency of the two conditions in

different age groups and partly to the progress of antituberculosis therapy.

SOOY.

**Evaluation of Laryngeal Nerve Paralysis, Particularly the Recurrent Laryngeal Nerve.**

*Takenaka, B., Yakata, K., Hara, Y., Yamawaki, K., Sugano and Kimura, N.: The Jour. of Otolaryngology* (Toyko) 26:1 (Feb.) 1954.

Takenata and his associates report their statistical study on 64 cases of laryngeal paralysis. Their investigation led them to believe that there are several points of doubt in the conventional concept of Semon-Rosenbach law. Among 64 patients whose records were studied, recurrent nerve paralysis was observed—40 in the left vocal cord, 11 on the right, and in three, both right and left cords were involved. They classified the position of the cords as cadaveric, midline and between these two positions according to their mirror examination. There were nine cases of paralysis of crico-arytenoideus lateralis and one of arytenoideus muscle. Of the various causes which brought on laryngeal paralysis, 17 were thought to be due to cervical adenitis and metastatic spreads from the distant organs, five thyroiditis, nine aortic aneurysm, six post-thyroidectomy, three pulmonary lesions, four mediastinal tumor, two polyneuritis, six influenza, one poison gas and nine unknown. Of the three cases of recurrent nerve paralysis which developed following thyroidectomy, the vocal cords assumed cadaveric position in two and a half way between a complete abductor paralysis and cadaveric position in another. In four, with mediastinal tumors, all their vocal cords were in cadaveric position.

They have also observed that paralysis of thyro-arytenoideus internus and that of crico-arytenoideus lateralis were caused by influenza, acute laryngitis and polyneuritis. They consider this form of paralysis a muscular disturbance rather than a recurrent laryngeal nerve disorder. Behavior of the vocal cords was different in different individuals with the same etiologic factors depending on the time of observation.

They used electromyograms from laryngeal muscles during the process of phonation and respiration and attempted to determine the part played by each muscle. According to these authors, their investigation prompted them to believe that the Semon-Rosenbach hypothesis requires a modification.

HARA.

**Laryngeal Stenosis of Tuberculous Origin.**

*De Villa-Sante, J. P.: Acta Otorinolaringol. Ibero-Amer.* 5:3:163-179, 1954.

A case of tuberculosis, with bilateral pulmonary cavities, developed pharyngeal and laryngeal involvement. Earache on swallowing, paresthesia, cough, pain in the left apex, severe pain in the throat, dyspnea and asphyxia were present. It was necessary to perform emergency tracheotomy. Laryngoscopic study revealed extensive ulceration of the supraglottic region and of the aryepiglottic folds with profuse exudation of yellowish mucus. The laryngeal symptoms responded to treatment of the general tuberculous condition. However, dysphonia persisted, due to the residual scar tissue.

In another case in which emergency tracheotomy was necessary, the dyspnea was complicated by cyanosis and anoxemia. Macroscopic examination revealed a tuberculoma of the right ventricular band, extending to the left side and covering an infiltrating edematous lesion which appeared to be malignant. There was no ulceration. Except for persistent bronchial catarrh, no significant etiologic factors were noted. The dyspnea, which at first appeared only on effort, became constant. A whistling sound accompanied respiration. The condition was diagnosed as miliary tuberculosis. Tuberculin, calcium and vitamin C were given. After scar tissue had formed in the laryngeal passages it was possible to remove the cannula. Subsequently the patient breathed without dyspnea. The vocal cords, visible only on the left side, were opaque and flaccid, probably due to slight arytenoid arthritis attributable to the earlier tuberculous perichondritis. Due to the sluggishness of the cords, dysphonia was present. Otherwise there was marked improvement.

Pulmonary tuberculosis with laryngeal complications is frequently fibrocaseous or nodular and productive, with or without cavitation. The exudative, ulcerous type, with profuse expectoration of infected matter is rare. The laryngeal lesions are generally on the same side as the pulmonary disease. For this reason therapeutic pneumothorax and phreniectomy benefit the laryngeal as well as the pulmonary condition.

Authorities disagree as to the advisability of biopsy to confirm the diagnosis, in view of the danger of causing ulceration and loss of tissue. Tracheotomy, total laryngectomy, laryngostomy, etc., have been found to cause excerbation of the tuberculous pulmonary disease and to induce bronchopneumonia because of the tracheal respiration and the retention of bronchial secretions. Tracheotomy is therefore indicated only when it is necessary to relieve progressive

asphyxia or spasm, in stenosis of the larynx with an unfavorable prognosis. Even in such cases the procedure may be fatal. However, the author considers it justified as an emergency measure in dyspnea. If the stenosis is permanent and the cannula cannot be removed, extirpation of the scar tissue is recommended.

The alarming dyspnea may be due to stricture of the lumen of the glottis or to obstruction by tumors or fungoid formations, or edema and inflammation caused by drastic therapy, such as cauterization. It may also be due to arthritic immobiliation of the cricoarytenoid articulation, or to paralysis of the dilator nerves.

Unless the stricture endangers the life of the patient, specific medical treatment with streptomycin and isonicotinic acid should first be employed. However, the streptomycin causes cicatrization of the tuberculous lesions and consequently retraction of the fibrous tissue and regression of the dyspnea, which necessitates tracheotomy. Not all forms of tuberculous laryngitis respond to streptomycin. The exudative types are generally benefitted. In the second case cited, no streptomycin was given, and the clinical improvement was due to the laryngeal rest afforded by tracheotomy, general rest and nutrition, and the favorable constitutional state of the patient.

HIGBEE.

## ESOPHAGUS

### The Effect of Cortisone on the Scarification in Corrosive Lesions of the Esophagus.

*Flabey, L. E. and Kock, H.: Acta-laryng.*, Suppl. 109, pp. 32-48.

Working with cats, circular lesions of the esophagus were created by using 30% NaOH applied by means of special instruments through the esophagoscope. Postmortem examinations with careful histological studies were carried out. The animals were grouped as follows:—Control, (no therapy), those receiving penicillin, those receiving cortisone, those receiving both penicillin and cortisone. Therapy was by the intramuscular route.

Penicillin was effective in inhibiting stricture formation by preventing secondary infection and granulations. Cortisone treatment was effective in the prevention of stricture formation through inhibition of fibroblastic activity. A combination of both penicillin and cortisone gave the best results.

HILL.

## MISCELLANEOUS

**Reflectory Swelling of Salivary Glands.**

*Strelka, Josef: Acta Otolaryngologica Cechoslovaca* 2:212 (Nov.) 1953.

Previously reported cases of sudden swelling of salivary glands following manipulation inside of the esophagus are reviewed and three cases reported. In all of the three cases the swelling was confined to the submaxillary glands and happened after esophagoscopy. In one case it was twelve days after a burn with alkali. In the second case it was after an esophagoscopy done in order to extract a foreign body stopped above a scar of many years' duration. In the third case it was a cardiospasm.

The swelling is explained as a reflex pattern with the afferent pathway in the vagus nerve and the efferent one in the chorda tympani and glossopharyngeal nerve.

SOBIESKI.

**An Experimental Study of the Small Blood Vessels of the Spiral Ligament and Stria Vascularis of Living Guinea Pigs During Anaphylaxis.**

*Weille, F. L., Martinez, D. E., Gargano, S. R., Irwin, J. W., and Gilchrist, Mary, Gordon, Priscilla, Technical Assistants. Laryngoscope* 64:8, 656-665 (Aug.) 1954.

Despite great technical difficulties these authors have been able to study the small blood vessels and blood flow of the stria vascularis and spiral ligament of living guinea pigs. The blood vessels were observed in over fifty preparations. No emboli or thrombi were observed prior to anaphylactic shock. Guinea pigs were sensitized to egg white and while under observation a shocking dose of antigen was given. During anaphylaxis the changes noted in the spiral ligament included constriction of arterioles and initial constriction followed by dilatation of venules.

When death was delayed for several minutes emboli and thrombi were observed in both arterioles and venules. After recovery from anaphylaxis most of the emboli disappeared but many of the thrombi in the vessels persisted.

SENTURIA.

**The Study of Epithelial Cilia and Sperm Flagella with the Electron Microscope.**

*Fawcett, D. W. Laryngoscope* 64:2. 557-567 (July) 1954.

In the past five years methods have been developed for embedding tissues in plastic and cutting sections less than a twentieth of a micron in thickness. This permits the preservation of normal spatial relationship of tissue components and the study of the fine architecture of cells. Using these methods a comparative study was undertaken of ciliated epithelia derived from an annelid worm, two varieties of mollusks, two species of amphibia and three different mammals, including man.

An interesting uniformity of internal ciliary structure was demonstrated among the species studied. A constant number of longitudinal filaments was found in all cilia and flagella. Two filaments were present in the center with nine closely joined pairs evenly spaced around them. The asymmetrical basal body and the central pair of filaments appeared to bear a constant relationship to the direction of beat. The ciliary rootlet fibers showed a distinct cross-banded structure.

This study once again demonstrates the amazing progress which is possible as new scientific "tools" are developed and utilized.

SENTURIA.

**Clinical Observations of the Facial Nerve in Chronic Purulent Otitis Media as an Early Alarm Signal.**

*Yannoulis, G. E.: Monatsschr. f. Ohrenheilk., Vienna, Austria,* 88:175, 1954.

The author describes a phenomenon believed never to have been published before, but repeatedly observed by him and E. Ruttin in some cases of chronic otitis media, never in acute otitis media.

The patient is asked to extend his head backward and to close his eyes. The closure of the *inner* canthus of the eye on the side of the diseased ear lags behind the outer canthus, or may even be absent.

Radical mastoidectomy proved erosions on the Fallopian canal in all cases of a positive phenomenon.

The phenomenon is explained by the anatomical situation of the nerve bundles which form the facial nerve: The fibres of the eye lie anteriorly and laterally and are therefore subject to damage or irritation in cases of local erosion of the facial nerve.

FISCHER.

**The Effect of Stellate Ganglion Block on Bell's Palsy.**

*Nakata, Tatsuo: The Jour. of Otolayng.* (Tokyo) 26:15 (Jan.) 1954.

There are three known approaches to the stellate ganglion. Nakata prefers the anterior route by which he introduces daily 10 cc of one per cent novocain solution for a period of ten days. In favorable cases he noticed improvement in the facial expression in five or six days after initiation of the therapy. This form of treatment was attempted on eight cases and there was only one failure. He states that chances of recovery from Bell's palsy are greatest if the blocking is performed within a week after onset of the illness.

HARA.

**Clinical Pharmacological Effects of Acth and Cortisone in the Treatment of Surgical Patients.**

*Kinsell, L.s Jour. Internatl. Coll. Surg.* 21:230 (Feb.) 1954.

The author reports on his experience with over 90 major general surgical problems in which ACTH and cortisone were administered postoperatively in conjunction with appropriate antibiotic therapy.

The results are summarized as follows:

1) Within two to six hours after institution of hormonal therapy significant improvement in general circulation was noted.

2) Decrease or disappearance of local and systemic inflammatory symptoms.

3) A "smoother" postoperative course than was anticipated.

4) No interference with wound healing was noted with the exception of two extremely debilitated and malnourished patients.

The author specifically cautions against protein tissue breakdown, potassium depletion, and sodium retention with the recommendation of high caloric diet, excess potassium and sodium restriction during therapy.

SOOY.

Problems of the Otolaryngologist in Surgery of the Neck.

*Gilbert, J. G. and Feit, L. J.: Jour. Internatl. Coll. Surg.* 21:93 (Jan.) 1954.

The authors present a simplified "working" classification of cervical lymph nodes, a variety of incisions for the surgical removal of

tumors of maxilla, lip, jaw, tonsil, tongue, pharyngeal and hypopharyngeal areas are clearly illustrated.

A brief outline of the surgical technique used with each completes the text.

Sooy.

**Surgery of Ozaena.**

*Lewkowitz, A.: Oto-Laryngologica Israeliana* 3:66, 1953.

The present status of surgery for atrophic rhinitis with ozena is concisely reviewed.

Wittmaach's "saliva therapy" consisting of implantation of Stenson's duct into the maxillary sinus is not recommended.

Paraffin injections and fat implantation are also decried.

The author lists eight of his patients in which implanted ivory was removed due to necrosis. Bone implantation, however, is felt to show promise but failures result from infection. Lautenschlaeger's method of medial mobilization of the lateral nasal wall is felt to assure a higher percentage of success than Hall's procedure which consists of medial mobilization of the medial wall of the antrum.

Hinsberg's "plate seam method" is felt to be the operation of choice since it moves the entire lateral nasal wall medially by virtue of section of the anterior buttress of the maxilla, and the "plate seam" which allows the shifted wall to remain stable.

The principal objection to this method is the implantation of silver plates, lead balls, and wire to maintain position.

The author avoids this objection by the use of a metal nasal splint clip which is applied through the bilateral gingivo-labial incisions after the lateral nasal wall has been mobilized and by bilateral compression maintains the apposition of both lateral nasal walls to the septum. This clip is removed on the eleventh post-operative day.

One of the chief post-operative complications, dacryocystitis, results from the necessity of transecting the bony and membranous naso-lacrimal duct in mobilizing the lateral nasal wall.

This occurred unilaterally in four out of 223 cases and was successfully treated by extirpation of the sac in two cases and Toti procedure in two cases.

Other complications include: Purulent sinusitis, 12 cases; otitis media, 1 case.

Results include lessening of turbinate atrophy, disappearance of foul secretion, odor and headache.

Sooy.

**Re-Evaluation of Air-Conditioning from the Point of View of Otolaryngology.**

*Kinkade, Joseph* M.: *A. M. A. Arch. Otolaryng.* 60:15-23 (July) 1954.

Air conditioning of homes and public places has been undertaken to establish optimal body comfort and maintain normal performance and efficiency under adverse atmospheric conditions. Thermal comfort is determined by such environmental factors as air temperature, relative air humidity, air movement and radiation of surrounding structures but is also affected by clothing, physiologic conditions, and the sensation of comfort is influenced by psychic reactions. Only a limited number of these factors can be influenced by air-conditioning.

Control of indoor temperature is one facet of air-conditioning. Insufficiently controlled indoor temperature causes steeply contrasting degrees of warmth at different room levels; under these conditions subjects with a tendency to vasomotor disturbance may experience attacks of vasomotor rhinitis and chilling of the feet may bring on a cold. Air-conditioning shock exists when there are pronounced temperature differences between controlled thermal environment indoors and the natural atmosphere outside. The difference in temperature levels may reach 20-30.° Rapid transition from one environment to the other imposes a burden on the heat-regulating mechanism of the body, which is well tolerated by healthy persons but individuals with a tendency to vasomotor rhinitis or hay fever adjust more slowly and show an increased susceptibility to infections, especially rhinitis and other upper respiratory infections. The effect of air-conditioning shock should be modified as much as possible by providing zones of intermediate temperature or by appropriate clothing. To avoid air-conditioning shock it is desirable to limit the difference between outside and inside dry-bulb temperatures to 5° F and to effect bodily comfort by changes in air humidity, whenever feasible.

Relative air humidity is important for the upper respiratory tract, although the human body can compensate for wide variations in relative humidity, but the optimum humidity lies between 45

and 50 per cent. Low relative humidity predisposes to respiratory diseases and nervous disorders. Patients with vasomotor rhinitis are most comfortable at a dry-bulb temperature of 70-72° F and relative humidity of ±55 per cent. During an acute attack of frontal sinusitis and other upper respiratory tract infections, a humid atmosphere produces more comfort, but when the condition clears a dryer environment is preferred. However, there is not agreement among all investigators on this point. Humidities of 15 per cent or less result in drying of the nasal mucosa.

Control of air motion, another aspect of air-conditiong, must be adjusted to the prevailing temperature, and should be higher in summer than in winter. Freedom from drafts will be of benefit to those suffering from ear, nose or throat diseases. Patients complaining of a stuffy nose desire air freshness, which is dependent on relative humidity, air movement, adequate exchange of air and radiation from surrounding surfaces. Atmospheric conditions which tend to reduce intranasal cooling increase vascular congestion, giving rise to sensations of stuffiness or actual nasal obstruction.

In a review of air sanitation, figures are presented which the author feels demonstrate the importance of chemical disinfection of recirculated air. An air-conditioning system provided with air washers and using an effective germicide will lower the indoor pathogen count. Some of the problems in the hygienic control of air within air-conditioned spaces are discussed and it is pointed out that many as yet await solution. It is here that the author feels the otorhinolaryngologist should work with the physiologist and bacteriologist in determining the hygienic requirements of the individuals occupying the air-conditioned space. The otorhinolaryngologist must study the working and living conditions of his patients and their reactions to this environment and thus provide the engineers with medical standards of air hygiene based upon the effect of air-conditioning on the patient with upper respiratory disease.

HILDING.

**Atypical Localization of Actinomycosis.**

*Ninger, Prof. F.: Ceskoslovenska Otolaryngologie* 2:195-197 (Nov.) 1953.

The author describes a case of a 32 year old male, who presented an abscess in front of the right auricle. Two months later he had an acute otitis media and mastoiditis, treated by mastoidectomy.

The wound of the mastoidectomy was slow in healing and a swelling appeared at the lateral cervical region. On opening this cervical abscess the actinomyces was found in the pus.

In spite of x-ray treatment and penicillin injections the patient presented a neuro-psychotic manifestation and was admitted in this condition to the department of the author.

An abscess in front of the superior cervical spine was found on x-ray films and lumbar puncture showed a purulent reaction. The patient died some days later and autopsy showed meningitis, frontal and occipital lobe abscesses, subcortical pulmonary abscesses, actinomycosis was obtained from these areas.

SOBIESKI.

# Books Received

**Segmental Anatomy of the Lungs.**

*By Edward A. Boyden, Ph.D. (Med. Sc.), Professor Emeritus of Anatomy, The Medical School, University of Minnesota.* Large 8 vo. cloth, 276 pp., 125 figs. and 12 plates. McGraw-Hill, N.Y., 1955. (Price $15.00)

A complete anatomical text on the lungs with special emphasis on structural minutiae so important to the surgeon, the bronchoscopist and the roentgenologist. The illustrations, many in color, are superb.

To quote the publisher, "it is the first comprehensive study to show the relation of arteries and veins to segmental and subsegmental bronchi and to employ a uniform system of naming and numbering related bronchi, arteries and veins. It is the first to extend analysis of bronchial and vascular structures to subsegmental levels. It is the only account based on first-hand knowledge of critical stages in the development of segments in human embryos."

**Reactions with Drug Therapy.**

*By Harry L. Alexander, M.D., Emeritus Professor of Clinical Medicine, Washington University School of Medicine, St. Louis, Missouri.* Cloth, 8 vo., 301 pages, 33 illustrations, W. B. Saunders Company, Philadelphia, 1955. (Price $7.50)

Alexander has assembled a comprehensive catalogue of drugs and their untoward reactions as they have been described in the literature of the past thirty years.

He remarks that although some 350,000 substances have been employed at some time as drugs, and that man may presumably be sensitized by any of them, a number estimated as less than a thousand are in common use and at most a few hundred of these have been demonstrated to induce hypersensitivity.

These he classifies, and the disturbances they cause are painstakingly described, together with the more acceptable types of treatment. All sources are given (a tremendous list of references) making this an important reference book for anyone concerned with the handling or administration of drugs.

**Diseases of the Nose and Throat in Children.**

*By T. G. Wilson, M.B., B.Ch., Litt.D., F.R.C.S.I., M.R.I.A., Ear, Nose and Throat Surgeon, Dr. Steevens' Hospital, National Children's Hospital, Dublin, etc., etc.* Large 8 vo., 307 pp., 145 illus. Grune & Stratton, N. Y., 1955, cloth.

This book, as the title indicates, deals with the special problems in the diagnosis and treatment of otolaryngological conditions in the young. The approach is practical, the text concise and the illustrations well chosen.

Especially valuable are the many references, thoroughly documented, to recent work in prophylaxis, virus diseases, and audiology. The role of the parent in the various situations receives its proper emphasis.

**Erlebtes aus der Thorax-Chirurgie (The Story of Thorax Surgery).**

*By Prof. Dr. Rudolf Nissen,* Basel (in German) paper, large 8 vo., 64 pp., 99 illustrations. George Thieme, Stuttgart, 1955. (Price: DM 14.40) ($3.40)

**Asthma Bronchiale**

*By Franz Wyss, Director of the Medical Clinic of the University of Bern,* 8 vo., paper, viii - 120 pp., illustrated, Georg Thieme Verlag, Stuttgart, (in German) distributed in the United States by the Intercontinental Medical Book Corporation of New York, price $4.00, 1955.

**Medical Progress—1955. A Review of Medical Advances During 1954.**

*Edited by Morris Fishbein, M.D.* Cloth, 8 vo., 346 pp. McGraw-Hill, N. Y., 1955. (Price $5.00)

# Notices

## SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

General announcements and invitations to participate in the Sixth International Congress of Otolaryngology to be held in Washington, D. C., May 5-10, 1957, will be mailed to all otolaryngologists throughout the world within the month.

The program of scientific papers will be high-lighted by invited speakers on three principal subjects to be presented before the Plenary Sessions. The subjects will be announced shortly.

A motion picture program, scientific and commercial exhibits and various social functions are being arranged.

## IV INTERNATIONAL CONGRESS

There are a few copies of the Proceedings of the 4th International Congress of Otolaryngology still available for purchase.

The publication contains all of the numerous papers, and is well produced. Copies may be had, at Five Guineas, from

The Secretary
British Medical Journal
B. M. A. House
Tavistock Square
London, W. C. 1.

## PORTUGESE OTORHINOLARYNGOLOGICAL SOCIETY

On March 20 the Sociedade Portuguesa de Otorrinolaringologia e de Bronco-esofogologia was founded, and held its first meeting in Lisbon under the presidency of Dr. Alberto Luís de Mendonça. The Secretary is Dr. António da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

## AMERICAN LARYNGOLOGICAL ASSOCIATION

### CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1956.

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

Dr. Edwin N. Broyles,
Editor Transactions
1100 North Charles St.
Baltimore 1, Md.

---

## UNIVERSITY OF PENNSYLVANIA

Announcement is made of the personal fundamental course in Bronchology, Esophagology, Gastroscopy and Laryngeal Surgery at the Graduate School of Medicine, University of Pennsylvania. The course begins September 8, and continues through September 19.

Courses are scheduled usually in January, June and September avoiding conflict with the meeting dates of the National Medical Societies.

Application may be made to Dr. Aims C. McGuinness, Dean, or to Dr. Gabriel Tucker, Chairman of the Department of Bronchology, Esophagology, and Laryngeal Surgery.

---

## TULANE UNIVERSITY OF LOUISIANA
## SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endoscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship.

An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

---

## ACADEMY HOME STUDY COURSES

The 1955-1956 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

---

## SECOND WORLD CONGRESS OF THE DEAF

The Second World Congress of the Deaf will take place in Zagreb, Yugoslavia, Aug. 23 to 27, 1955. Elaborate scientific and social programs are being arranged.

For information address Organizing Committee, Palmoliceva No. 4, Zagreb, Yugoslavia.

---

## UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 19 through October 1, 1955. This Assembly will consist of two parts.

Part I—September 19 through September 24, 1955, will be devoted to surgical anatomy of the head and neck, fundamental prin-

ciples of neck surgery and histopathology of the ear, nose and throat. This week will be under the personal direction of Maurice F. Snitman, M.D.

Part II—September 26 through October 1, 1955, will be devoted entirely to lectures and panel discussion of advancements in otolaryngology. The chairman of this section will be Emanuel M. Skolnik, M.D.

Registration is optional for one or both weeks.

* * *

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

---

## TEMPLE UNIVERSITY

Temple University offers a postgraduate course in bronchoesophagology October 17-28, 1955 and a postgraduate course in laryngology and laryngeal surgery September 19-30, 1955.

These courses are to be given in the Department of Laryngology and Broncho-esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is $250.00. For further information please apply to Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

---

## BACK COPIES WANTED

The management of the ANNALS desires to buy, at $1.50 each, copies of the following numbers which are out of print:

March, 1951
March, 1950
March, 1949

Please address communications and journals to the BUSINESS OFFICE, P. O. Box 1345, Central Station 1, St. Louis, Mo.

# OFFICERS

OF THE

## NATIONAL OTOLARYNGOLOGICAL SOCIETIES

---

### American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, New York.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

### American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Next examination: Palmer House, Chicago, October 3-7, 1955.

### American Broncho-Esophagological Association

President: Dr. Daniel S. Cunning, 115 E. 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: Mount Royal Hotel, Montreal, May 15-16, 1956.

### American Laryngological Association

President: Dr. Bernard J. McMahon, St. Louis.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Seigniory Club, Montreal, May, 1956.

### American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Dean M. Lierle, Iowa City.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, May, 1956.

### American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Francis W. Davison, Danville, Pa.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

### American Otological Society

President: William J. McNally, M.D., 1509 Sherbrooke Street West, Montreal 25, Quebec, Canada.
Secretary-Treasurer: Lawrence R. Boies, M.D., University of Minnesota Hospitals, Minneapolis 14, Minnesota.
Meeting: Seigniory Club, Montreal, Quebec, Canada, May 11-12, 1956.

## The American Society of Ophthalmologic and Otolaryngologic Allergy

President: F. Lambert McGannon, Lakewood, O.
Secretary-Treasurer: Michael H. Barone, M.D., Buffalo, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

## VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
Secretary-General: Paul H. Holinger, M.D., 700 North Michigan Ave., Chicago 11, Ill., U. S. A.
Meeting: Washington, D. C., May 5-10, 1957.

## International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: IV International Congress, Buenos Aires, October 28-29, 1955.

## Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan M. Tato, Buenos Aires.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
President of the Congress: Dr. J. H. Font, Puerto Rico.
Secretary of the Congress: Dr. C. E. Muñoz MacCormick, Santurce 29, Puerto Rico.
Meeting: San Juan, April 8-12, 1956.

## Pacific Coast Oto-Ophthalmological Society

President: Leland G. Hunnicutt, M.D., 98 N. Madison Ave., Pasadena 1, Calif.
Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.

## Canadian Otolaryngological Society

President: Dr. G. Edward Tremble, Montreal, Can.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario, Can
Meeting: Lake of Bays, Muscoka, Ont., June 16-18, 1955.